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ANNALS OF
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TRAUMATIC NEUROSES IN COURT *

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I. INTRODUCTION

WE can think of no more vexed or vexatious law-medicine problem than the proper appraisal and just compensation of so-called "traumatic neurosis." [1] Our intent is not to hack and hew, first with the scientific sword

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Because of lack of space, it has been necessary to omit most of the numerous legal citations with which the manuscript was documented. Readers wishing to consult the original sources will find these references published in full in Virginia Law Review, 1943, xxx, 87.

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[1] As the Supreme Court of Washington said, "An allowance of damages in the cases of traumatic neurasthenia touches the border of speculation at best." Mickelson v. Fischer, 81 Wash. 423, 142 Pac. 1160 (1914).

Although medical literature has not neglected traumatic neurosis, very little seems to have been written regarding its legal implications. See TIBBITS, F. V. W.: Neurasthenia, the result of nervous shock, as a ground for damages, Cent. Law Jr., 1904, lix, 83. Some of the medical material is outmoded and many of the cases cited involve nervous shock, a physiological response, rather than traumatic neurosis which is a psychological reaction.

More recently, Stotter has put by question, without attempting to answer it, one of the salient legal problems posed by neurotic responses, namely: "In negligence cases, does mental pain and anguish include unconscious exaggeration of pain and all the host of mental ills that are often precipitated by any traumatic event cast upon one that is predisposed to mental illness? . . . Whether such mental pain and incapacity to function is the proximate result of a specific act on the part of someone else is quite hard to answer. There is always back of these cases a pre-disposition, a preëxisting cause. In the words of the psychiatrist such would be termed only a precipitating cause, real causes going back often to childhood experiences. The legal definition of proximate cause helps us but little and would no doubt be broad enough to cover what is meant in psychiatry by a precipitating cause." Having cracked the lid of this Pandora's box, the author closes it quickly, leaving it for others to open it full wide. He does not attempt to gather or to analyze the authorities.

STOTTER, R. O.: Extent of liability for injuries to neurotic person yet to be decided, The State Bar Jr., Calif., 1941, xvi, 44.

The authors have published a version of this study for the legal profession buttressed with numerous citations of cases and certain special appendices which would only encumber a *medical* presentation. See Virginia Law Review, 1943, xxx, 87.

and then with the legal axe. We shall attempt, with some doubts, the more delicate and difficult task of weaving scientific and legal threads into a garment, albeit a Joseph's coat, which jurist, trial lawyer and expert witness may all wear with some degree of satisfaction.

A person who seeks compensation for traumatic neurosis must find some foundation for liability, and his legal claim will fall into one of the following categories:

1. A *tort* action for personal injury allegedly inflicted by the fault (negligent or intentional) of the defendant;
2. A *workmen's compensation claim* for disability allegedly due to an accidental injury received in the course and scope of employment;
3. A *war risk insurance claim* for total and permanent disability allegedly incurred before the insurance policy lapsed for non-payment of premiums;
4. A claim under a *life, health or accident insurance policy* by a person in civilian life.

II. General Clinical Characterization of the Neuroses

In a consideration of disease entities, one hopes to have a knowledge of etiology, pathology, or pathological physiology, a clear description of the symptoms and course of the disorder. These factors in complete form are not available in regard to those disorders which we designate as "psychoneuroses."

In a discussion of medical problems, as of all scientific matters, a clear definition of terms is usually a prerequisite. Unfortunately, it is not possible to define precisely the term "psychoneurosis." In fact, most textbooks baldly state that one can describe the symptoms of the psychoneuroses, divide them into symptomatic categories, but that one cannot either limit the territory of the disorders or tell where the psychoneurosis is to be separated from the normal. Such statements refer, naturally, to the borderline case, not to the full blown conditions of which we shall speak. Nor is it difficult for the experienced psychiatric examiner to spot the usual pre-neurotic constitution, if proper heed be paid to the individual's history and behavior patterns rather than solely to a search for an organic disease or lesion. It is fundamental to our concept that no structural pathologic lesion of the nervous or visceral systems has been discovered or indeed exists to explain the psychoneuroses. This viewpoint has certain consequences. It places the psychoneuroses in a different compartment from most diseases; it does not allow for a check of diagnosis through studies of pathology. A second fundamental concept is that the disorder or disorders are chiefly manifest in the mental life of the sufferer and, therefore, largely subjective, although many of the symptoms have organic or objective components. This second principle has led to the conception of the etiology of the psychoneuroses as in the field of the psyche, and one usually considers the cause as psychogenic.

Summing up the foregoing, it may be said that the psychoneuroses represent a group of symptom complexes or syndromes having no structural lesion, arising psychogenetically or out of the stresses and experiences of life, and manifested largely by subjective feelings and thoughts with some secondary physiological deviations. One must also assume that some individuals are more able and some less able to withstand the stresses and vicissitudes of life. And so the concept of individual vulnerability is introduced, with the further implication of the stability of the constitutional equipment. This brings in another term difficult to define and to measure, but a factor which must be estimated and assigned a value, as we shall see later. One may well believe that no one can withstand certain severe stresses, and therefore vulnerability is a matter of degree of resistance. This means we must take the resistance of the average person, roughly estimated, as our norm in determining whether the supposedly causal stimulus would have been adequate to produce neurosis in an ordinary individual. Undoubtedly, training or conditioning increases or decreases the threshold of vulnerability. Or, one may even reverse the point of view and see in the psychoneurotic an individual with a talent or capacity for worry, fear, concern, and other emotional and thinking mechanisms, possibly paralleling the function of imagination, which can be developed to a very high degree. Whatever the point of view, it is necessary in our opinion to take into account the pre-traumatic personality for the purpose of discovering what part of the total injury really represents a preëxisting neurotic constitution merely expressing itself by more obvious symptoms in response to stimuli which would cause no such symptoms in a normally constituted person. The legal import of this concept is enormous. It means that persons who develop more patent forms of neurosis in response to traumatic stimuli inadequate so to injure a normal person, are not caused thereby to develop the neurosis as a new and original condition. It is legally erroneous and socially unjust to compensate them on any such theory. Such cases are properly to be regarded as instances of aggravation of preëxisting injuries or impairments, and so compensated modestly. The neurotic constitution is the major factor in the disability, and it antedates the particular exacerbation of symptoms for which the plaintiff seeks damages.

It may also be accepted as true that all individuals have some tendency or capacity to worry, to be fearful, to be concerned, and that all have some psychoneurotic manifestations. This is much like saying that no one is free of some fear of bodily disease, but not thereby ill. As psychoneurotic mechanisms are well-nigh universal, one needs some rule of thumb for making a diagnosis and evaluating the case under consideration. It is a general working principle that when symptoms impede the efficiency of an individual or make life pretty uncomfortable, the diagnosis may be justified. It must be remembered that there need be no parallelism between the severity of symptoms and the effectiveness of the individual. Many of the most efficient executives, researchers, teachers, physicians, and lawyers have marked psycho-

neurotic phobias, anxiety states, compulsions, and other symptoms. Nor is it possible to evaluate the amount of discomfort such symptoms engender. In fact, there is often a suspicion that they give satisfaction, if not pleasure. And in the literature and clinic, attention is given to the presumptive protective value of the symptoms, their use for gaining some purposeful end, or their value as safety valves.

As the result of contributions to the study of the psychoneuroses by Janet, and especially by Freud, it is generally conceded that the cause of psychoneurosis lies in emotional and mental conflicts which have not been adequately faced or resolved, and which have been pushed aside from the normal thinking processes of the individual. The distress caused by the inability properly to resolve such conflicts is supposed to give rise to various types of discomfort, and thus the symptoms represent a diffuse form of expression. Whereas this is the point of view held by the majority of students of this subject, there is another school of thought which sees in the psychoneurotic an individual with an organic defect in the form of a poorly knit or poorly constructed nervous system. The latter point of view lacks sufficient scientific substantiation and for this reason, at the present time, has relatively little support, although lip service is generally given to the statement that if and when more information comes to hand, the more psychological view may be modified.

As already stated, it is the usual custom to describe rather than define the psychoneuroses, and for purposes of exposition, this group of disorders is usually sub-divided into three or four categories, namely, hysteria, neurasthenia, anxiety states, and the compulsive, obsessive states. These categories are by no means mutually exclusive, but, on the contrary, the symptoms characteristic of each category are likely to be found in greater or less extent in a case falling in any group.

1. Characterization of *hysteria.*

The main characteristic of hysteria is that symptoms are referable to the sensory-motor nervous system. Thus, the patient will present symptoms of paralysis, that is motor weakness, or changes in sensation. The hysterical patient may present paralysis of an extremity, of half of the body, or he may be unable to phonate and talk only in a whisper, or he may be unable to talk at all (aphasia). Convulsions similar to those seen in epilepsy may occur. In the sensory sphere, one meets patients showing areas of anesthesia, that is, inability to appreciate touch, pain, or temperature changes; total blindness, limitation of the field of vision, or double vision. The patient may be completely deaf, or show disorders of other special senses. Loss of memory (amnesia), double personalities, and symptoms of this type, are also classified as hysterical. In other words, those disorders not due to real changes within the nervous system, but giving symptoms similar to those found in true organic disease apparently arising from psychologic causes, form the group designated as hysteria.

2. Characterization of *neurasthenia.*

The neurasthenic group has as its main symptoms the complaint of weakness, sense of exhaustion, various pains, aches, and distress of a bodily nature, associated, as a rule, with concern about the functions of various organs. Frequently in cases of this group, one finds digestive distress, attacks of diarrhea, pain in the bladder with frequent desire to micturate, a feeling of pressure in the chest with concern about the state of the heart, headaches with fear of brain tumor, difficulty in concentration, disturbed sleep frequently resulting in a concern about sanity. With the preoccupation of the sufferer with his bodily sensations and functions, the pleasure in living is greatly reduced and a sense of futility and lack of interest occurs.

3. Characterization of *anxiety states.*

In the anxiety states, one finds an individual who becomes extremely panicky on occasions, usually without any understanding of the reason for this panic. These attacks are likely to be of relatively short duration, but represent real states of tremendous agitation. Such states of necessity are accompanied by the physical component of acute fright, namely, rapid pulse, strongly beating heart, tremors, cold perspiration, and a sense of impending collapse. These physical signs, which are part of fear and panic, naturally lead the patient to have concern about his viscera.

4. Characterization of *obsessive compulsive syndrome.*

The obsessive compulsive syndrome is manifested by the patient having the idea that he is compelled or is likely to be compelled to carry out acts which are contrary to his ordinary desires. Thus, in the compulsive syndrome, the individual may be greatly distressed because he feels that he will throw himself out of a window, that he will harm his child, that he will run some one down by automobile. The obsessive states are those in which the individual has thoughts running through his mind which he feels unable to control or put aside. Sometimes these will be in the form of indecent expressions or licentious ideas and mind pictures. The individual will usually say that it is much like a tune running through one's mind of which one cannot get rid. Because of unpleasant connotations, those thoughts or pictures interrupt the ordinary flow of thinking and become extremely distressing. In this category one also usually considers the phobias or fears, such as the fear of crossing the street or being in closed places, the fear of riding on a street car or train, or going more than a short distance from home, the fear of high places. These fears, which the individual will agree are unreasonable and contrary to his better judgment, nevertheless lead to a sense of panic if he tries to do the thing which produces the fear.[2]

[2] P, a 23 year old graduate of McGill University in electrical engineering, decided to gain experience by working for a time as a lineman. D's employees failed to cut off the power from a line on which he was working. An electrical current leaped with a spark or a flash from a wire 4 inches away, which carried 16,000 volts, and went through his body, entering at one of his hands, and going out at one of his feet. It was not possible to show how much electricity actually leaped from the high tension line, but apparently he was not so much injured as terrified. P developed mixed symptoms of neurasthenia and hysteria,

As one might anticipate, the compulsive syndrome is rarely the basis of claim in traumatic neurosis, but obsessive states about the permanence of disability and fears for family are commonly linked with general anxiety reactions in the neuroses of injured workmen.

The reader will have observed that all these symptoms and symptom-complexes, except those of the hysterical states, are primarily subjective. It is what the individual thinks and feels or fears that is important. In so far as these subjective feelings interfere with normal living, working, and playing, in so far are they serious and disabling. The objective evaluation can be made only by watching the behavior of the individual. If the phobiac never leaves his room because of the state of fear and panic such an attempt produces, one must conclude that it has disturbed the entire tenor of his life.

Necessity for Distinguishing Residual Symptoms of Traumatic Injury to the Brain from Similar Symptoms of Psychoneuroses.

Accepting the foregoing postulates concerning the psychoneuroses, it follows that symptoms arising as the result of organic brain injury due to trauma are not properly considered as psychoneuroses. Whether the traumatic brain injury is in the form of cerebral concussion, hemorrhage, or contusion does not alter this statement. Therefore, disorders of this type are not in the scope of this discussion, except in the form of differential diagnosis in order that they may be excluded.

Symptoms arising from traumatic brain damage often have a remarkable similarity to those of a true psychoneurosis, and are distinguishable chiefly by objective evidences of real organic brain changes. Such evidences are protracted periods of unconsciousness; blood in the cerebrospinal fluid and increased intracranial pressure, both discoverable by the virtually riskless diagnostic expedient of lumbar puncture; changes in the reflexes; motor weakness and sensory changes of specific neurological variety; and mental changes that can be directly associated with disorder of brain structure. In many instances, the decision as to whether symptoms are the result of traumatic effort or are of a psychoneurotic nature is a matter of judgment rather than a matter of definite objective differentiation. In addition, it frequently happens that there is a combination of the effects of organic brain disturbances and psychoneurotic symptomatology. The latter may at times mask the former.

As the effects of traumatic injury of the brain recede, psychoneurotic manifestations may take the foreground among the presenting symptoms. These factors produce difficult practical problems of decision, but do not affect the underlying theoretical concepts about the psychoneuroses.

If one accepts the point of view that the psychoneuroses are of purely psychological cause and nature, and if they are to be considered as the result

in part manifested by temporary paralysis followed by involuntary twitching and shaking of his muscles, and by a deep rooted dread and fear of electricity which presented a serious obstacle to pursuit of his chosen profession. Verdict and judgment for P for $7,500, affirmed on appeal. Summerskill v. Vermont Power and Mfg. Co., 91 Vt. 251, 99 Atl. 1017 (1917).

of unresolved conflicts, it is in order to ask how a trauma to the head or some other part of the body can be causally related to the appearance of neurotic symptoms. Obviously, the relationship is not a direct one, and is not dependent upon the force or nature of the physical impact, but rather, any relationship present is an indirect one in which the trauma affords the optimum conditions for the outbreak of symptoms.

Perhaps this can best be illustrated by reference to the war neuroses, as is essentially described in the literature of both World Wars. Following partial burial or concussive effects of near-by explosions, many individuals developed psychoneurotic states of the various sub-divisions of this disorder. The formulation runs somewhat as follows. A soldier, worried, anxious, frightened, and fatigued by the conditions of warfare, is faced by the conflict between the instinct of self-preservation and the desire to do his job faithfully and well. A minor concussion or other accident following an explosion leading to removal to a medical installation affords an opportunity through unconscious mechanisms for the development of symptoms, the continuance of which lead to removal from the front and care in hospital. Thus, the symptoms solve the man's immediate problem. It is assumed that this does not occur through conscious planning but rather occurs in part spontaneously, and in part by suggestion, and therefore cannot be considered as malingering. The accident or trauma could then be considered as the opportune moment for the development of symptoms which the psychological conflicts and the personality structure of the individual had made possible. A similar mechanism can be readily conceived as operating in the case of superficial traumatic injury sustained in civilian life, substituting only other forms of conflict and discontent for those existing in the war situation. Among the conflicting emotions and desires, fears and worries, and anxieties existing in ordinary civilian circumstances, one considers discontent with a job, fear of losing one's income, unsatisfactory marital relations, lack of good social adjustments, and almost any variety of maladjustment. An accident, whether it be to the head or other part of the body, may then break down the individual's resistance and allow the psychoneurotic mechanisms to have full sway. When an injury to the head has occurred, several factors arise which in themselves are important in the causation and prolongation of symptoms. For example, an individual may be fearful that a blow to his head has damaged his brain, and the result of such fear, with the associated idea of being unable to work and support himself and his family, can be productive of symptoms. Discontent with the type of job at which the accident occurred, fear of discharge if he returns to work and is not entirely competent either because of general inability or because of lessened capacity resulting from the injury may cause psychoneurotic manifestations.

The symptoms arising in this fashion, associated as they so frequently are with the question of compensation, often evoke the suspicion that the injured individual is consciously producing or exaggerating his symptoms for

financial gain. As already frequently stated in this discussion, there is no method of accurately measuring the intensity of the complaints described by the injured person. These facts raise special problems of evaluation in fixing a just compensation, in cases where it is due, and we shall advert to them at a subsequent point in this paper.

III. Specific Law-Medicine Problems Presented by Traumatic Neurosis

A. *The Law of Torts. Assuming there has been an impact produced by defendant, to what extent shall the law protect the idiosyncratic or excessively vulnerable person in allowing him damages?*

Assume for the moment that study of the claimant's pre-traumatic personality shows a neurotic constitution or condition sufficient to make the subject an extreme reactor to stimuli which would only mildly affect the average person, if at all. In his case a $5.00 touch may become a $10,000 disability. Shall the law protect this fragile fellow to the full, or where shall legal and social policy draw the lines limiting the compensation he may obtain?

This is one of the most intriguing problems to be found in the realm of law, for it plumbs legal theory and the philosophy of social justice. One is aided in coming to just conclusions by remembering the essential ingredients of the trial formula:

$$\text{Duty} \xrightarrow{+} \underset{\text{(Plaintiff or claimant must prove)}}{\text{Dereliction}} \xrightarrow{+} \text{Proximate Causation} \xrightarrow{+} \text{Injury} \;\Big\|\; \leftarrow \underset{\text{must prove)}}{\text{Defenses (Defendant}}$$

Any plaintiff or claimant who seeks money compensation for alleged injury, in order to establish a prima facie case of liability adequate to get his claim to the jury or to sustain a verdict, has the burden of proof to show by substantial evidence the concurrent existence of duty, dereliction, direct causation and damages. If he fails to do so, the court, on motion timely made by the defendant, must instruct the jury to return a verdict for the defendant. The limits of liability for injuries ascribed to psychic stimuli have been discussed elsewhere.[3] It seems clear that a defendant commits no dereliction if he indulges conduct which would not injure a normally constituted person, even though it actually does harm an idiosyncratic individual, P (plaintiff). This rule is subject to the assumption that the actor cannot be charged with notice of the unusual risk and does not intend or desire to injure.[3] Here D escapes liability on the ground that P cannot prove his conduct culpable, and this negatives any primary liability.

In the majority of traumatic neurosis cases, there is usually some minor impact and superficial injury, inadequate to cause neurosis on a traumatic

[3] Smith, H. W., and Cobb, S.: Tort liability for psychic stimuli, in Smith on Scientific Proof and Relations of Law and Medicine, 1944, Matthew-Bender and Co., Albany, N. Y., Vol. 1 (in press).

basis, but sufficient to found a primary liability. The question is not now one of culpability or no culpability, liability or not, but whether, and in what way, idiosyncrasy may be proved by a defendant to limit the damages he must pay. Both British [4] and American courts,[5] with a singular unanimity,

[4] This doctrine was expressed in Dulieu v. White & Sons (Eng.), 2 K.B. 669 (1901), 70 L.J. (1901) K.B. Div. 837, one of the historic precedents on legal liability for psychic stimuli. P alleged that on July 20, 1900, she was pregnant but was working behind the bar of her husband's public house, when D negligently drove a pair horse van into the tavern allegedly frightening her so badly that she suffered nervous shock and a consequent miscarriage. Justice Kennedy in ruling on a demurrer held the injury actionable. He rejected defendant's contention that the plaintiff's unknown vulnerability would defeat liability, saying: "It may be admitted that the plaintiff, as regards the personal injuries, would not have suffered exactly as she did, and probably not to the same extent as she did, if she had not been pregnant at the time; and no doubt the driver of the defendant's horses could not anticipate that she was in this condition. But what does that fact matter? If a man is negligently run over or otherwise negligently injured, it is no answer to the sufferer's claim for damages that he would have suffered less injury, or no injury at all, if he had not had an unusually thin skull or an unusually weak heart."

In the *Dulieu* case, the conduct was negligent when tested by risk of injury to an average person, and accordingly the question was not one of primary liability, but whether P's unknown vulnerability would limit the damages recoverable. In Owens v. Liverpool Corp. (Eng.), 1 K.B. 394 (1937), MacKinnon, L. J. failed to perceive this crucial distinction. He seemed willing to apply the doctrine to raise a primary liability for a purely idiosyncratic response. This was an extension not warranted by British precedents or by prevailing concepts of tort law as to what constitutes culpable conduct sufficient to make a defendant liable.

[5] In American law two cases may be regarded as "stem" authorities. In each case it was clear from the facts and the court's doctrinal approach that defendant's conduct was such as to create a primary liability to a plaintiff possessed of average health. The vital holding was that the defendant could not have his damages reduced by proving that plaintiff's unknown idiosyncrasy caused his injury to exceed that which a person possessed of average health would have sustained.

Purcell v. St. Paul City R. Co., 48 Minn. 134, 50 N.W. 1034 (1892), 16 L.R.A. 203; Spade v. Lynn & Boston Railroad, 172 Mass. 488, 52 N.E. 747 (1899), 43 L.R.A. 832, 70 Am. St. Rep. 298, 5 Am. Neg. Rep. 367, 11 A.L.R. 1124. Such is the rule generally followed by American jurisdictions. The main difference in judicial approach lies in the varying vigilance which courts show in excluding preëxisting impairments from compensation. Authorities are collected in the Digest System under Damages, Keys 33, 95, 132(3), 168, and 208(2).

What part of plaintiff's injury represents compensable aggravation, and what part non-compensable poor health antedating the accident requires most careful discrimination by jury and judge if awards are to be kept at just levels.

Flood v. Smith, 126 Conn. 644, 13 A.(2d) 677 (1940), illustrates this problem of separating non-compensable from compensable factors. As the result of an automobile collision caused by negligence of D, P_1 and P_2 sustained injuries more excessive than an average person would have suffered from a like stimulus. Proof showed that two years before, P_1, a 28 year old man, was hurt in an automobile accident, suffering a fracture of the skull and injury of his nervous system, from which he made a substantial interim recovery. P_2, his companion, was a 70 year old library cataloguer. Her medical history revealed that prior to the accident, she had undergone two nervous breakdowns and the surgical removal of a cancerous breast. In the instant accident P_1 and P_2 sustained bruises and suffered extreme nervous shock, in P_2's case aggravated by her morbid fears that a blow received at the site of her amputated breast would reactivate her cancer. The jury awarded P_1 $3,500 and P_2 $4,000. The trial court thought these damages were excessive. It therefore granted D's motion for a new trial unless P_1, by remittitur, should relinquish $1,275 of the verdict, and P_2 $2,027. P_1 and P_2 appealed. Held: The damages awarded by the jury were not excessive. Case remanded with orders for trial court to enter judgment for the full verdicts. The Connecticut Supreme Court said, "The plaintiffs are entitled to recover full compensation for all damage proximately resulting from the defendant's negligence, even though their injuries are more serious than they would otherwise have been because of preëxisting physical or nervous conditions." This statement of legal doctrine expresses the majority view, but it glosses over the clinical certainty in such cases that part of the post-accident symptoms are due to pre-accident causes and do not represent aggravation by the accident.

have held that once a defendant can be culpably connected with the general type of injury he has caused the plaintiff to suffer, the bars are down. The defendant cannot then be heard to say: "I should not be held to pay for the full harm but only for the degree of injury which a normally constituted person would suffer." The concept is sometimes pithily stated that a tortfeasor takes his victim as he finds him. As between the culpable actor and the innocent subject who is injured, there is equity in placing the loss on the former.

Over enthusiasm for this cliche has led some courts to the erroneous conclusion that once culpability can be proved so as to create a primary right of action, the defendant is liable for all consequences actually caused by his wrongful conduct. The better view, however, is that remoteness of expectation of injury runs both to culpability and to compensation. There is an independent doctrine of remoteness of damages, whose exact limits are still in process of being fixed. Its cardinal principle is that no harm actually suffered is compensable if it falls outside the risk of the defendant's conduct. Damages may be remote for three reasons:

(1) Following upon defendant's conduct there may be a new and intervening cause which stands in closer (or, as some say, more proximate) relationship to the final injury.[6] This involves a defect in proof of one term of the liability formula.

[6] Assume that D's conduct results in a superficial physical injury to P. D cannot be held liable in damages for independently caused sequelae, as, for instance:

(1) *If subsequent to the accident, P begins worrying about what might have happened to him and in that event to his family, thereby developing an anxiety neurosis.*

In Phelps Dodge Corporation v. Industrial Commission, 46 Ariz. 162, 49 P.(2d) 391 (1935), the facts were that X, a miner, ran 500 feet to reach fresh air after blast of a "missed hole" caused shafts and drifts of the sulphide ore mine to become filled up with smoke, sulphur gas and dust. He suffered no injury but thereafter developed a neurosis from brooding over what might have happened to him, and in that event what would have been the lot of his family. Held, since the neurosis was not caused by nervous shock produced by the episode, but by subsequent brooding, the neurosis was not result of an injury sustained by accidental means, and was not compensable under terms of the Arizona Compensation Act.

(2) *If as the result of such brooding P is led to commit suicide:*

In certain interesting cases claim has been made under Workmen's Compensation Acts for suicide due to insanity allegedly caused by an accidental injury received in course of employment. Courts have held it is not enough that insanity was indirectly caused by the injury if the more immediate cause was worrying and fear of losing employment (Grime v. Fletcher (Eng. 1915), 1 King's Bench 734; 8 B.W.C.C. 11, C.A.) or depression and brooding over inability to work (Withers v. London, Brighton and South Coast Rail Co. (Eng. 1916), 2 King's Bench 772, 9 B.W.C.C. 616, C.A.). So, also, there is a defect in causal connection where decedent's preparation showed a "moderately intelligent mental power which knew the purpose and physical effect of the suicidal act." Kazazian v. Segan, 14 N.J. Misc. 78, 182 Atl. 351 (1938).

In the type case mentioned under (1) and (2) supra, there may be *actual causation*, but it must be admitted that lapse of time, the entry of independent causes, and difficulties of proof, justify the law in drawing a boundary line of liability, though somewhat arbitrarily, to mark off and exclude the morass of speculation.

(3) *Where the disability has arisen from anxiety, worry or brooding over the proceedings for compensation, or has arisen from a cause unrelated to the accident itself, there is no right of compensation.*

A mental state or nervous disturbance caused merely by pendency of compensation proceedings is even less necessarily referable to the injury.

(2) The injury, in type, would be remote in expectation of the prudent actor.[7] This involves a defect in proof of one term of the liability formula, namely *dereliction.*

(3) The particular injury may not be remote in expectation, and no logical reason may exist for its exclusion under the four terms of the liability formula, yet courts may deny compensation on grounds of extrinsic policy. Such considerations constitute arbitrary restrictions on the damages recoverable. Usually they find their justification in difficulties of proof, or in judicial aversion to making the load of liability inordinately heavy.[8]

Coffey v. Coffey Laundries, Inc., 108 Conn. 493, 143 Atl. 880 (1928).

Where the accidental injury involves a substantial stimulus which directly causes the neurosis, without delay, the case is different: here there is no defect in causation and no question about liability. Thus, where claimant developed an anxiety neurosis due to fear and anxiety about a hole in his skull produced by injury and operation, this neurosis was properly held to be compensable. National Lumber & Creosoting Co. v. Kelly, —— Colo. ——, 75 P.(2d) 144 (1938).

Deliberate and unnecessary exposure of oneself to injury, even if remotely occasioned by D's conduct, may constitute the immediate cause of harm suffered and bar recovery of damages.

D, excavating adjoining land, impaired the lateral support of P's hotel. P was warned that guests should be cleared from the imperiled wing. P did this, and after waiting for two hours, labored strenuously, and unnecessarily, in helping to move her furniture out. She alleged that fright drove her to this overexertion and that the latter caused a nervous breakdown. Verdict and judgment for P for $2,000. On appeal, reversed: there was a break in proximate causation, for P did not act spontaneously in response to fright or to escape an immediate emergency. Her injury was caused by her independent, deliberate acts.

Cherry v. General Petroleum Corporation, 172 Wash. 688, 21 P(2d) 520 (1933).

[7] Here the injury is outside the risk of defendant's conduct. Assume that D, hurrying to a foot-ball game through a large crowd, carelessly brushes P aside, the impact being enough to inflict a superficial wound. P is a hemophiliac and bleeds to death from a scratch thus received. Or suppose P has the unusual disease *fragilitas ossium,* a rare congenital weakness of bones characterized by multiple fractures which occur spontaneously or upon moderate jolting. As a result of D's impact, P's bones break in several places. In these cases D may be held responsible for nominal damages if he is negligent, but he can hardly be taxed with liability for the full injury. This is true because the result is not a mere exaggeration of a foreseeable harm, but belongs to a category of consequences entirely beyond any foreseeable risk of injury involved in D's conduct.

Again, suppose that D negligently collides with P's car, so wounding P that the latter must go into hospital for treatment. Without any notice of their incompetency, P accepts treatment from the hospital physicians, and suffers further injury as a result of their negligent ministrations. P can hold D liable in damages for both his original injury and for the effects of the malpractice.

The authorities are collected in 8 A.L.R. 506, 39 A.L.R. 1268, and 126 A.L.R. 912.

Assume instead, that P's further injury is not due to medical malpractice but to lightning striking the ward in which he is confined. P cannot hold D in damages for this additional injury. D's conduct in injuring P created a new risk that the latter would come under medical care and have his wounds aggravated by negligent treatment. That D's conduct was calculated to send P into the hospital did not, however, involve a foreseeable risk that P would be struck by lightning, nor did it increase the probability of such an injury.

In Denison, B. & N.O.R. Co. v. Barry, 98 Tex. 248, 82 S.W. 5 (1904) reversing (Tex. Civ. App.) 80 S.W. 634 (1904), defendant railroad negligently constructed a dump in such way as to throw water back on P's home. P's wife was pregnant. As the water rose up to the floor of the house she became frightened at the prospect of drowning and fled with her husband to a safe place. This episode caused P's wife to suffer a threatened miscarriage involving pain and sickness of several weeks' duration. D was unaware that P's wife was pregnant. The Texas Supreme Court in reforming a judgment obtained by P in the trial court, upheld the sum awarded for property damage, but denied any right for compensation for the wife's physical illness on the ground that this injury was too remote a consequence to be anticipated by the actor.

[8] One who causes a violent collision can foresee that a scene of horror will be created which may cause involved persons such as the plaintiff, who behold the spectacle, to suffer

We may hope and expect to see fuller and more effective use made of this doctrine of remoteness of damages. A bold court would be warranted in holding that the development of neurosis following a minimal impact is an idiosyncratic response, in which cause-effect relationships are hopelessly obscured, and that difficulties of proof, appraisal and just compensation are so great as to raise an extrinsic policy against their redress.[9] All the logical grounds for holding a particular item of damages remote, while recognizing a primary liability for the impact, and any nervous shock, could be validly invoked.

Courts timid about thus cutting off entirely the liability for neurosis caused by stimuli too insubstantial and inadequate to so injure the average person, may limit the measure of damages by resorting to another principle. It is the rule everywhere that a plaintiff afflicted with a preëxisting illness or impairment, cannot hold one who injures him liable in damages for his prior condition, but only for its aggravation.[10] It is good logic and settled law that a defendant is not liable for an injury, or any part thereof, which he did not cause. Now traumatic neurosis cases yield nicely to separation into three classes:

(1) A very large group where a stimulus, psychic or traumatic, patently inadequate so to affect an average person, causes the plaintiff to develop neurosis. These cases are all presumptively instances of an idiosyncratic response. They bespeak a pre-neurotic personality, and a competent psychiatrist could usually expose evidence of definite personality neurosis antedating the allegedly causal episode.

nervous shock. Liability exists for nervous shock or injury due to such immediate psychic stimuli.

The defendant also can foresee that persons going away from the scene of a railroad wreck may suffer secondary shock from beholding the wounds of fellow passengers, but damages are not allowed for this more distant consequence.

Nor will the courts allow damages for nervous shock and consequent injury caused in a parent, not present within the circle of risk, but induced by secondary reports that her child has been injured or killed through defendant's negligence.

So, too, it is a most natural and foreseeable consequence of injuring a minor, that his parents will suffer mental anguish and nervous shock from discovering his injury or maimed condition, or through sympathy for his deformity, but the law refuses to hold the defendant answerable in damages for such results.

A pregnant woman who is injured through defendant's negligence may recover damages for mental anguish due to her reasonable fears that the child will be born deformed, but if she suffers a traumatic miscarriage she can recover no damages for death of the child, or for mental anguish caused thereby; nor can she recover damages for mental anguish caused from beholding traumatic deformities in a living child after it is born.

[9] In particular cases, these considerations may warrant a refusal to recognize any *actual causation*, especially if the stimulus is trivial and the symptom-free time interval long. Stanford v. Longe & Wolfe (La. App.), 199 So. 608 (1941); The Rigel (Eng.-Admiralty) (1912) P. 99. And see Cookson v. Barber Co., 120 Me. 527, 115 Atl. 285 (1921).

An able neuropsychiatrist of our acquaintance went so far as to say it would be a signal service if courts could be induced to deny compensation in *all* cases of traumatic neurosis, as this would do more than any medical means to banish the disorder. We would not go so far as to espouse universal denial of compensation, but certainly this point of view has much in its favor in regard to neuroses which appear following trivial stimuli.

[10] This rule is axiomatic. Defendant is entitled to have the jury instructed, in proper cases, that it is for them to consider plaintiff's previous physical condition in determining whether P's nervous symptoms were or were not consequences of general ill health.

(2) At the other extreme we have a smaller group of cases, where the plaintiff has sustained severe associated injuries or is led by mode of treatment to the reasonable belief that he has a serious and permanent disability, or is subjected to a harrowing psychic stimulus of a kind not readily effaced from the mind. The development of neurosis in these cases is not presumptively idiosyncratic, but each case must be judged on its own merit after proper medical examination.

(3) A middle group, seemingly the smallest of all, contains border-line cases falling between groups (1) and (2). This group tends to diminish in size as we gain increasing insight into the neuroses and as the cases investigated are studied by competent psychiatrists.

Thus, though we do not know all there is to be learned about the neuroses, it is feasible, with a nearer approach to exact justice, to sort the cases into these three main groups for purposes of making a just appraisal and evaluation of a particular case. We have applied this technic in surveying all the traumatic neurosis cases, as far as we could find them, which have reached appeal courts of the British Empire and of the United States. We find that three-fourths of them fall without question in the group of idiosyncratic response to trivial stimuli. Yet it is precisely in this group that lack of proper information causes juries and courts to allow extravagant awards.

This disturbing phenomenon would soon be corrected if triers of fact and appeal judges realized that the neurotic who presents a deplorable spectacle in court, was not immediately before the accident truly "as fit as a fiddle" or "the strongest woman in the house" or "the picture of health." A plaintiff can always bring trusting neighbors to court bearing witness to his hale and hearty pre-accident health, and ready to describe calamitous changes noticed in him shortly after receipt of the trivial impact. These laymen see only the flowering stalk of the neurosis, not the extensive roots underground, nor how the trivial stimulus, like a little rain water, combines, with the sun's warmth and other environmental factors, to cause rapid surface growth. Actually, as we shall continue to stress, the trivial stimulus which causes appearance of new symptoms (or the flowering stalk of the neurosis) merely adds to a process already underway.

If we may shift metaphors, another fit analogy is to compare the waxing of this type of traumatic neurosis to the breaking open of an old scar, which has healed, only to break open at a later date in response to some trivial stress or some purposeful need of the individual.

Still again, we may compare the pre-traumatic condition of such a neurotic to a cracked vase. The unobservant or untrained eye may not notice the crack, but only that the vase will hold water. It is only when the crack spreads and the vase will no longer hold water that he is conscious of any defect. But the law of torts must follow the rule of the market place and take cognizance that a cracked vase is not so valuable as an intact one. If the defendant's conduct causes the crack to spread, he may justly insist that he shall not make compensation on any assumption that the vase was previ-

ously perfect. So here, traumatic neurosis resulting from trivial stimuli should be treated as mere accession to, or aggravation of, a preëxisting impairment.[11] Once this truth is grasped we can expect to see the measure of damages lowered to more modest levels.

IV. Characterization of Traumatic Neurosis Litigation

Who are the plaintiffs and who the defendants in this type of litigation? In analyzing those cases of traumatic neurosis which reached the appeal courts of the British Empire and of the United States, we divided them into two main classes as follows:

A. Cases of traumatic neurosis following trivial impact or psychic stimuli[12];

B. Cases of traumatic neurosis incident to serious physical injuries.

We found that less than 3/8 of all the appeal cases fell in group B, despite the great numbers of serious personal injuries which fill the courts in modern times. Doubtless some of the 3/8 group were not neurosis cases but examples of neurotic-like symptoms following actual organic injury to the brain. Thus, more than 5/8 of all cases fell in group A. This conforms with clinical observations that most neurosis occurs on a "neurotic" or purely psychological basis.

In group B, involving traumatic neurosis incident to serious physical injuries, 43.5 per cent of cases were males, and 56.5 per cent females. In group A, containing traumatic neurosis following trivial impact or psychic stimuli, 27.1 per cent of cases were males and 72.9 per cent females. This finding further accentuates basic differences between the two groups of cases, and throws light upon the psychological mechanisms involved. Males venture into places of peril as much as females and so are as frequently exposed to the group A stimuli. But the male is usually the breadwinner; his thoughts are distracted from his experience by the tasks of his job, and further, he has much to lose and little to gain by developing a neurosis. The female is usually at home, has more time to ponder upon the experience, and more to gain and less to lose from developing symptoms. The independent post-accident psychological forces conducing to neurosis are apt to be more potent in her case.

It is noteworthy that more than 90 per cent of the defendants are healthy corporations, for the most part sturdy public utilities symbolic of wealth and

[11] This principle is so fundamental that it is above the vagaries of conflicting evidence, or defects in technical proof: we regard it as a scientific axiom entitled to recognition under the doctrine of judicial notice.

Some courts already have progressed part way to this goal, in reducing verdicts as excessive where the jury has allowed full damages despite evidence of plaintiff's preexisting neurotic state or symptoms.

[12] Of 129 cases in 13 only could we say that the stimulus was such that it *might* have sufficed to cause neurosis in an average person.

Of these 13 cases, in three the neurosis was not due to the initial stimulus but to fears of permanent injury aroused by the manner in which the attending physician treated the case.

the ability to pay. The purposeful mechanisms of neurosis may operate on a sub-conscious level, but the hope of being compensated thrives best when a stout corporation can be looked to as the party defendant. There is reason to believe that one does not develop a neurosis so readily if the wrong is done by one's neighbor.

We were particularly impressed by the inadequacy of the stimulus in most of the group A cases (5/8 of all traumatic neurosis) to cause a neurosis in an average person. This finding substantiated our impressions that in most cases of traumatic neurosis, the alleged stimulus is only a trigger mechanism, and not the substantial cause of the neurosis.

Lastly, our analysis disclosed that group A cases of pure neurosis are being compensated almost as liberally as the group B cases. In the group A cases, the average jury verdict was for damages of $8,317.20, and the average judgment entered by the appeal court was for $6,037.29. In the group B cases, the average verdict awarded was for $9,655.96, and the average judgment entered by the appeal court amounted to $8,058.72. That the same range of compensation should be awarded in the two series is startling when we consider that the group B cases involve serious physical injuries, often permanent injury to the brain. Our finding in this respect shows that juries and courts have an exaggerated notion of the nature and permanence of traumatic neurosis following upon trivial stimuli.

V. Applied Problems of Proof in Arriving at a Just Result in Traumatic Neurosis Cases

Medical science recognizes that genuine traumatic neurosis can occur, that it can cause disability through psychological mechanisms which lead the subject to fixed beliefs that his health is impaired, and that it follows symptom patterns which permit diagnosis. British and American courts have accepted the medical concept that traumatic neurosis is a real injury, and so prima facie compensable in damages if the claimant further proves existence of duty, dereliction and direct causation. The vexatious problem has been how to appraise causation and how to arrive at a fair award of damages. There is no single cause for all the symptoms of such a neurosis and the sorting out of compensable injury is obscured by a variety of circumstances. We intend to explore the pervasive problems and the special ones which arise in the litigation of:

a. Tort actions based on ordinary negligence;
b. Workmen's Compensation cases;
c. War Risk Insurance cases.

A. *Tort Litigation.*

1. The Dramatic Appeal.

Psychological reactions aroused in the trier of fact play a dominant rôle in the making of proof. Traumatic neurosis, with its absence of organic

injury, might engender distrust and skepticism leading jurors to return small verdicts. On the other hand, the pitiable appearance of certain neurotics conduces to arouse sympathy and to swell damages. A female plaintiff is more able to capitalize on such sympathy reactions than a male.[13] For a proper female the court room may become a special stage for the enactment of a drama calculated to overthrow jury conservatism. Examples might be multiplied, but Judge Ulman's description of trial of such a case in his court illustrates at once the force and tempo which such a presentation may attain and the impact it may produce upon an attentive jury.[14]

Case Summary. Plaintiff, an actress known on the vaudeville and concert stage as "The Sweet Singer of the South," was playing an engagement in a Baltimore theater in the year 1928. Through negligence of defendant's stage hands in manipulating the "scrim-drop," a curtain made of tightly stretched gauze and weighted at the bottom with lengths of iron pipe, it became fouled and ripped open, releasing a long length of pipe. The pipe toppled toward plaintiff, and though a fellow actor caught the main weight on his arm, the upper end of the pipe in falling to the floor brushed by plaintiff's head and neck and struck her on one ankle. No one could say with assurance that it actually did come in contact with her head.

Plaintiff swooned and almost fell, was carried into the dressing room where she remained in a comatose condition for half an hour and then was taken to her hotel and put to bed. A physician who then examined her could find no evidence that P had been struck or injured about her head or neck. He did observe that P was extremely nervous and that she complained of great pain in those regions. This physician saw her daily until she departed for New York toward the end of the same week. According to the evidence, P became progressively worse, lost her ability to speak, and became paralyzed on her right side. She returned to her home in Alabama where she was completely bed-ridden for two months and thereafter used crutches to accomplish limited locomotion.

In the meantime, P had filed suit against the theater and the case came on for trial 18 months after the date of the initial episode. P's father, a Southern gentleman, a judge from Alabama, recounted impressively the story of his daughter's girlhood, her education at school and college and her vocal studies in New York. Kodak pictures were offered in evidence, showing P as she appeared before the accident, a wholesome-looking girl standing under the trees on the lawn of her Southern home. Other witnesses described her vocal studies and professional success, imparting to the attentive jurors "a picture of a beautiful young woman, with a lovely voice, a great career in concert and opera opening before her, and an assured income of about twelve thousand dollars a year." At this juncture, two days after the trial began, P was called to the stand. "With difficulty she rose from her seat. Her dignified, elderly father held her on one side, her aunt on the other. They placed her crutches beneath her arms. With their help she walked slowly across the court room, dragging her right leg, and sank, exhausted, into the witness chair." P was a prize exhibit in her own behalf; she appeared on the stand as a hopeless invalid, much aged in appearance, with professional hopes and normal life blasted. In testifying, her voice would fade into a whisper after the utterance of a few words. When this occurred, P would move her left hand gently to the right side of her throat, and press firmly against it as though to move some obstruction inside. "Then she paused a moment; and when she began to speak, her voice was strong and vibrant. But only for a few

[13] In the cases studied juries awarded males verdicts averaging $7,198.23; in comparison, they awarded females verdicts averaging $8,801.09.

[14] Ulman, J. A.: A judge takes the stand, 1933, Alfred A. Knopf, New York.

seconds; and the painful cycle was repeated. Among other things, she told the jury about the phonograph records she had made and sold to one of the great companies that market them."

To demonstrate the character of P's voice as it was before the accident, P's attorneys were permitted to play in the court room a phonograph record she had made. "I was never more surprised. The selection was a sentimental ballad of the music halls. The phonograph was a cheap one. I expected to hear that half throaty, half nasal voice which one associates with the vaudeville stage. Instead, the softer parts of the song were produced in a sweet, round voice, full of gentle sentiment; and the high notes were sung *bravura* in a manner that would reflect credit upon some of the best song recitalists I have ever heard. The court room was tense. As the notes of the song rang out full and clear, the plaintiff sat huddled in the witness chair with her handkerchief to her eyes, weeping silently. Then she left the stand on her crutches, dragging her right leg. She was still weeping when she took her seat on the front bench beside her dignified old father from Alabama." Medical experts called by plaintiff and by defendant agreed that P had no organic injury, that her complaints manifested traumatic neurosis and existed only on a psychological basis. D's witnesses related how closest physical examination by specialists showed no physical disease. It was on prognosis that medical opinion diverged sharply: P's witnesses thought that termination of the litigation would help her condition somewhat but that she would never be able to face an audience and sing again from a concert stage. D's witnesses foresaw a complete cure in six months or a year and no reason why she should not get well enough to continue her career as though nothing had interrupted it.

"When the case was tried, the plaintiff had lost already about eighteen months from her work. Before the accident she had earned about $1,000 a month. Her medical and nursing expenses had amounted to nearly $2,000. So that was about $20,000 to start on. And the jury was instructed that it might allow for that vague something called pain and suffering and for its estimate of the losses which the plaintiff might continue to endure as a result of the accident." The jury, on June 7, 1929, returned a verdict in P's favor for $50,000, and in July, the defense dismissed its proposed appeal and entered into a compromise settlement for $40,000.

What was the subsequent medical course of this shattered plaintiff? The rose regained its petals, as can be seen by an extract from a college magazine published in Alabama in February, 1930. "Huntsville friends and music lovers were given a rare treat in January, when E. S. sang for the Art League at the Federation Club House. Music critics have pronounced Miss S's voice richer and better than ever, following the long rest she has had since an accident on an Eastern stage, when a curtain drop fell and seriously injured her. For the summer of 1930 she plans to go to Europe to continue her studies under famous continental teachers. Her charming personality, as well as her lovely voice, makes many friends for her. She is one of the most popular and sought-for singers of the time, and justly so."

This case illustrates several truths about traumatic neurosis: the patient is never as badly off as he appears; the symptoms often lend themselves to dramatic presentation in court; the trier of fact is apt to gain the false impression that the plaintiff has sustained a calamitous permanent disability, and that the injury is entirely ascribable to the defendant's fault. It demonstrates how the stresses, strains and excitement of litigation exacerbate symptoms, causing them on trial to appear more severe than they are. It shows also, how rapid and unexpected may be the complete cure derived from a "green-back plaster" in the form of a compromise settlement. Loss of past and future earnings is always material in fixing compensation, but the same

dramatic triumph may be achieved by a young lady from the 5 and 10 cent store,[15] or by a domestic, provided she has competent directors and producers.

The reader must not assume that we have any animus against traumatic neurosis; we would be the first to proclaim its reality and to dispute those who think that all such cases involve frank malingering. On the other hand, we agree with the court which astutely divined that traumatic neurosis is a condition *sui generis*. So true is this that traumatic neurosis cannot be compensated scientifically on any naive all-or-none theory of causation. Perhaps this warrants our reviewing distorted and distorting notions and factors, to the end of stating certain fundamentals which may help trial lawyer and judge in disposing of such cases.

1. *The plaintiff must be required to establish that he is suffering from traumatic neurosis.* One or two symptoms or findings will not suffice: exaggerated reflexes, for instance, may be found in many situations and do not warrant the diagnosis. Symptoms of pain, headache, dizziness and the like may be due, in whole or in part, to preëxisting illness, not neurotic in character, or to previous major operations, or to hormonal imbalance due to the menopause in a woman between 40 and 50 years of age. In some cases it is possible to show that the plaintiff had a full-blown neurosis before occurrence of the episode asserted to have caused it, in which event juries allow little or no compensation.

The plaintiff must be able to trace the onset of symptoms, with proper time relations, to a focal experience, traumatic or psychic, and to show a clinically accepted progression pattern, important requirements which we shall elaborate later.

There is no doubt that some general practitioners of medicine, or careless witnesses, are prone to fasten the diagnosis of "traumatic neurosis" to a collection of miscellaneous symptoms, many of long standing. Such diagnostic errors lead of course to unjust enrichment of the plaintiff by "sweetening the verdict," and the defendant is thus made to pay for preëxisting poor health which he did not cause. Courts, where new practice and procedure acts authorize the step, should appoint competent, impartial psychiatrists to examine and appraise cases of alleged traumatic neurosis.

2. *Medical examiner and lawyer must determine whether the traumatic neurosis in question represents the more common example of aggravation of preëxisting neurotic impairment or one of the rarer cases of traumatic neurosis arising de novo in a person of average constitution.*

[15] Kress v. Sharp, 156 Miss. 693, 126 So. 650 (1930), 68 A.L.R. 167. (P, a clerk in D's department store, was carrying four dozen bath towels in her arms in course of replenishing a counter. The steps she was descending were narrow and poorly lighted. She fell sideways, bruising her hip and back. After resting, she resumed her place at work, but did not continue, at the noon hour going home on the bus and walking part of the way. She had no physical injuries, but went to bed for six weeks, was treated by chiropractors for two years, and at the time of trial, some two years after the accident, was allegedly suffering from *hysteria* with intermixed compulsions and obsessions. Verdict and judgment for P for $25,000; on appeal, held excessive; judgment reversed and case remanded for new trial on limited issue of damages.)

A. *The Preëxisting Impairment Cases.*

This task of sorting cases is not so difficult as one might believe. The analysis naturally starts with a full description of the episode and surrounding circumstances alleged to have set off the neurosis. Those extrinsic circumstances which operate independently, and with which the defendant cannot be causally connected, must be excluded. We find that 5/8 or more of the court cases carry their own calling card in that the allegedly causal stimulus is obviously inadequate to affect a person of average nervous and psychic constitution. This fact is usually discernible by a layman, though medical testimony may help. Mind you, we do not say that in 5/8 of cases, there is no *actual causation* of neurotic symptoms, but rather that the average person would not be thus affected, so that appearance of traumatic neurosis is presumptively due to aggravation of previous impairment. In such cases the compensation must be modest, fractional rather than total.

We may demonstrate this group series of cases (the 5/8 group) by a few examples which recur constantly in litigation:

(1) *Common carrier cases:* The train on which plaintiff, a male passenger, was riding ran into the caboose of a freight train. The impact was not great. P, who was then sitting in the smoking car, was thrown forward and struck his head and shoulder lightly against the seat ahead. He sustained no physical injury and continued his business trip for several days, noting advent of mild symptoms referable to his head and neck. Gradually, most diverse symptoms involving every part of his body developed in a rich profusion characteristic of traumatic neurasthenia. P recovered verdict and judgment for $16,000, and on appeal the award was affirmed.[16]

(2) P, a 44 year old exporter weighing 230 pounds, was sitting at his desk in D's building when an area of plaster 2 feet square fell from the ceiling 10 feet above the floor. It flaked into fine particles as it fell. P either was not struck, and was merely frightened by the noise, or the impact was too slight to inflict physical injury. P testified that he was stunned, and that 15 minutes later he became very nervous. He went to several doctors, but refused to be hospitalized or to undergo lumbar puncture as an aid to diagnosis. The last specialist he consulted saw him 200 times in the course of a year. His pulse rate was accelerated to 90 (normal, c. 70), he had tremors, hyperactive reflexes, was apprehensive, lost weight and showed various symptoms of anxiety neurosis and hysteria. P recovered verdict and judgment[17] for $3,850, which on appeal was reduced to $1,000.

In these cases of neurosis following trivial stimuli, a competent psychiatrist usually can find tell-tale confirmation of preëxisting impairment, by close investigation of the plaintiff's past history. However, the inadequacy of the stimulus above should be recognized as sufficient basis for a psychiatric opinion that the particular case is one of idiosyncratic response involving aggravation of a pre-traumatic neurotic trend. It is no answer that plaintiff was previously able to work, as the two facts are not incompatible. Furthermore, ability to work is only one factor entering into the measure of damages in tort actions for personal injury.

[16] St. Louis, I.M. & S.R.R. v. Osborne, 95 Ark. 310, 129 S.W. 537 (1910).
[17] Klein v. Medical Bldg. Realty Co. (La. App.), 147 So. 122 (1933).

B. *Cases where there is no presumption against causal connection between stimulus and neurosis for the reason that the stimulus is of the substantial variety which might cause neurosis in a person of average constitution.*

In this category we place cases of traumatic neurosis incident to severe personal injury, and to psychic stimuli of very harrowing variety. Each case must be closely analyzed, as we cannot draw a generalization that presence of serious injury excludes the possibility that the individual already had a neurotic constitution. The pre-traumatic personality must be studied avidly, in the manner we shall suggest in discussing the Workmen's Compensation cases. Still, this group is sharply demarcated from the group A cases in that here the stimulus is regarded as adequate to produce a neurosis *de novo*. In serious head injury cases, as we have observed, the symptoms may be due to organic injury of the nervous system, to superimposed neurosis, or to both. Less serious injury to the head or to the back, or to other regions of the body, may result in neurosis if medical treatment is conducted in such way as to create fears in the patient that he has suffered serious or irreparable injury.

As we have hinted, though we regard barrel A as containing "bad apples," we do not believe that every apple in barrel B is a sound one. For instance, what more vivid laboratory could we have than war itself? Every soldier on the firing line experiences fear for his life, and may see the most cruel and abhorrent spectacles, psychic stimuli rarely duplicated in civilian life. Yet it is significant that aside from transient nervous shock, which is a *physiological* response,[18] only a small percentage of soldiers develop neurosis, a *psychological* response. There is accumulating evidence that a good portion of these had sub-normal resistance and prior impairment which should have been detected at the induction center by appropriate psychiatric examination. In any event, group B cases give us a residue of traumatic neurosis claims which may prove in the particular case, after adequate study, to merit 100 per cent compensation.

[18] SMITH, H. W., and COBB, S.: Legal liability for psychic stimuli, to be published in Virginia Law Review (March, 1944) and in SMITH: Scientific proof and relations of law and medicine, 1944, Matthew Bender & Co., Albany, N. Y., vol. 1 (in press).

Traumatic neurosis developing in soldiers at the front is best known to laymen under the inaccurate term "shell-shock." Readers will appreciate that this term hardly touches the essential cause-effect mechanisms involved. There are many misconceptions about the subject. See, for example, PAINTON, F. C.: There is no such thing as shell shock, The Readers Digest, 1943, xliii, 59. The title is catchy but unfortunately the statement it contains is erroneous. It is true that many soldiers exposed to heavy action develop nervous shock, a transient physiological state which may not progress to neurosis if they are kept near the front, treated by sedatives and rest to overcome psychological tensions and fatigue, and are gradually put back into action. The effort here is to prevent the flowering of neurosis by intercepting psychological elaborations. In the last war when such soldiers were invalided to base hospitals and treated as serious injuries with opportunity to brood and meditate, a larger percentage developed neurosis. Treatment near the front enables restoration of many soldiers to useful service who would have been disabled through neurosis under old methods of management. However, it is erroneous to assume that the shift in methodology represents an over-night discovery, for many of the lessons were learned from experience in the preceding World War (1914–1919), and this change in therapy has received attention in psychiatric literature for some years past. See MILLER, E.: The neuroses in war, 1940, The Macmillan Co., New York. KARDINER, A.: The traumatic neuroses of war, National Research Council, Washington, D. C., 1941.

3. *Nervous shock must not be confused with traumatic neurosis.*

As Smith and Cobb state,[19] nervous shock from psychic stimuli produces only relatively transient upset or disability through excessive physiological responses, except where the stimuli have a continuing force or repetitive operation, or where such responses cause injury by acting upon a pre-existing state of vulnerability.[20] Nervous shock per se, no more than trauma, produces traumatic neurosis, for that sequel is not a physiological but a psychological response. The recipient of the trauma or psychic stimulus reacts to it as a *focal experience* or organization point for his neurotic symptoms. Courts err when they fail to perceive this distinction and treat nervous shock and traumatic neurosis as the same phenomenon. Many persons will suffer transient nervous shock from psychic stimuli such as great fear, who will never progress to the development of a traumatic neurosis. Conversely, some persons who sustain no immediate nervous shock will begin to develop neurosis a few hours or a few days after the *focal experience,* thus illustrating that independent forces and more devious mechanisms are involved in the appearance of neurosis.

Example: P, a maid in the house of X, was engaged to N, an interned German (first world war). D, a private detective, desired to examine letters in X's house to determine whether or not they were forged, as he suspected. He said he was from Scotland Yard, representing the military authorities, and that he was looking for a woman who had been corresponding with a German spy. He hoped by this threat to induce P to give him access to the letters. P was frightened, went to the police at 9:00 p.m., and cried five minutes before she could tell her story; then sat on the stairs of her residence from midnight until 5:00 a.m. with a police whistle in her hand, without sleeping. She claimed that this experience caused her to develop neurasthenia. The evidence showed that previously P had been in a state of psychological turmoil from continued protests of relatives against her maintaining contacts with the German, N. P's physicians admitted on cross-examination that her neurotic symptoms might as likely be due to anxiety about N as to the episode mentioned. This would have rendered proof of causation of the neurosis ambiguous, had the court not held that P's account of her nervous shock corroborated the theory that her neurosis was the result of her fright. P recovered verdict and judgment for 250 pounds and this was affirmed.[21]

On the principles we have mentioned, we would say that P's immediate responses were compatible either with fright and transient nervous shock or preëxisting neurosis, whereas proof that the episode actually produced P's neurasthenia was conjectural.

4. *In the usual case of traumatic neurosis, symptoms are multiplied and exaggerated as a result of independent causes, and the patient's plight appears to be worse than it is.*

[19] Smith, H. W., and Cobb, S., *op. cit. supra* f.n. 52.

[20] For instance in more than 25 per cent of the litigated cases of alleged injury due to fright, the described harm was miscarriage of a pregnant woman. The authors mention such conditions as angina pectoris, a heart disease in which injurious or fatal attacks may be precipitated by excessive emotional stimuli. They also list diseases for which there is clinical evidence that psychic stimuli may precipitate or aggravate an attack.

[21] Janvier v. Sweeney (Eng.), 2 K.B. (1919) 316, 9 B.R.C. 579, 88 L.J.K.B. N.S. 1231, 63 Sol. Jo. 430.

Persons in group A (trivial stimulus cases) are almost always very suggestible. They can be led to believe that they must have struck their bodies on the cross bar of a street car seat when no such bar or obstruction was there. Their symptoms are greatly aggravated by the anxiety and excitement of litigation, a fact which the defendant is entitled to prove. These apprehensions about the oncoming trial are due partially to fears that their claim of injury will be disbelieved or held in contempt for want of objective lesions. Also, the self-serving mechanisms involved in neurosis invariably cause some degree of unconscious exaggeration or malingering in respect to symptoms, as the neurotic desires to be believed and wants his complaints to be convincing. Suggestions made by relatives and lawyers and the continuance of disability payments are additional extrinsic factors which cause neurotic symptoms to be aggravated or exaggerated. Thus the neurosis is not so bad as it seems, and the conclusion of litigation will usually cause many of the symptoms engendered by it to disappear. In our opinion 20 per cent to 60 per cent of the disability can be safely assigned to these extrinsic factors and expected to vanish upon settlement of the case.

5. *One must bear in mind that 12 to 36 months between stimulus and trial is a not unusual time interval, due to congestion of the courts, and that part of the neurotic symptoms may be traceable to independent, post-accident causes.*[22]

6. *To prove that the stimulus (traumatic or psychic) created by defendant actually caused neurotic symptoms, it must be shown that some or all of the characteristic symptoms appeared within a reasonable time after receipt of the stimulus.*

A vulnerable person may begin brooding a long while after an accident regarding what might have happened to him and in that event to his family, with the result that a late anxiety neurosis develops which should not be compensable. Causation is obscure enough, at the best, in traumatic neurosis cases, and proof of causal connection becomes too conjectural to be trusted, when a symptom-free period of more than a few hours, or at most of a few days, separates stimulus and onset of the neurosis. The plaintiff must be required to prove satisfactory bridging symptoms to connect stimulus and late neurosis, such as speedy and persistent complaints of pain, substantial nervous shock, or prompt and continuing symptoms showing altered psychological behavior.

[22] The facts of Hunter v. Fleming (Mo. App.), 7 S.W.(2d) 749 (1938), illustrate this possibility. On June 29, 1925, P, a married woman, was with her husband in the family car when it stopped dead on a street car track. D's conductor could see the stalled automobile 200 feet away but he continued to approach at a speed of 12 to 15 m.p.h. P screamed and waved her arms, but a slight collision occurred. This modest impact caused P to sustain superficial head injuries and a few bruises but no objective injuries of any consequence. Thereafter P developed nervous symptoms consistent with *neurasthenia* but proof of cause-effect relationships revealed that on Jan. 28, 1927, P had suffered a miscarriage. The examining physician attributed to this latter cause part of the symptoms of which P complained at the time of trial.

Thus defendant's counsel must make close inquiry into plaintiff's post-accident medical history to see if other illnesses or accidents have occurred which might account for some or all of P's symptoms or disability.

7. Traumatic neurosis cannot be regarded as a permanent disability.

Many courts have upheld excessive awards for traumatic neurosis because of their mistaken impression that the condition involves permanent disability. This is perhaps the most common misconception which now distorts the calculation of a just compensation. Many medical witnesses contribute to this misapprehension by testifying loosely that the condition "is of uncertain or indefinite duration." [23] We do not have adequate medical grounds to warrant such a prognosis. There is extremely good evidence that the average case of traumatic neurosis recovers within three to five years or even less, following the time a lump sum settlement is effected or the litigation is terminated.[24] There may be rare exceptions of a certain amount of irreversible injury due to the atrophy of disuse, if a hysterical contracture persists long enough for these secondary dangers to occur.

Some courts, in deciding that the traumatic neurosis is probably a permanent rather than a temporary disability, make capital of the fact that the neurosis has continued for a year or two between injury and date of trial, and without improvement. The inference is attractive, but hardly trustworthy, as pendency of a claim for compensation operates in a potent way to keep the neurosis in full bloom.

Testimony that traumatic neurosis may lead to brain abscess shows crass ignorance or deliberate imposition on the part of a medical witness, for brain abscess is due to infection, and the risk of this is not increased unless it be shown that the traumatic wound caused infection which then reached the brain. Even in appropriate cases, such a complication would occur or not within days or weeks of the injury. Another claim sometimes made by medical witnesses is that the neurosis may pass into psychosis, that is to say, frank insanity. Some courts seem to think that neuroses and psychoses are brothers and sisters. Such a relationship is not proved, nor indeed credited: we have no scientific proof that neurosis is a step on the way to psychosis.[25]

[23] This practice is a widespread and pernicious habit among expert witnesses; it lays a false foundation for a judicial inference that the disorder is a permanent disability.

[24] If anything, this allowance errs on the side of liberality. It is intended as an outside limit, for most cases recover more speedily.

[25] Judicial skepticism has kept some courts on the right path in this matter. In Louisville & N.R. Co. v. Creighton, 106 Ky. 42, 50 S.W. 227 (1899), P, a 38 year old woman, had received injuries in trying unsuccessfully to rescue a three year old child from the path of an oncoming train. Later she developed hysteria, and on the trial her physician predicted that this might progress to insanity. The jury awarded her a verdict of $17,500, but on appeal a judgment for this amount was reversed as excessive, the court pointing out that P seemed to be in possession of all her faculties and "she testifies . . . very lucidly in this case."

In Friedman v. United Rys. Co. of St. Louis, 293 Mo. 235, 238 S.W. 1074 (1922), P was motoring with her husband when their automobile was involved in a collision with D's street car. P was rendered hysterical but not unconscious by the impact, and en route to hospital in the car of witness Woody, P exclaimed to her husband, H: "Oh, daddy, you have killed me," to which H replied: "It was your fault, sweetheart, you grabbed the wheel." Thereupon, P replied: "I know it was, I don't blame you, sweetheart." This testimony was objected to on trial on the ground that P's hysteria rendered her mentally incompetent, but the trial court admitted the evidence as an admission against interest and the jury returned a verdict in D's favor. Held, on appeal: affirmed.

Courts are concerned about the possibility that an injured person may be imposed upon in the making of a compromise settlement. One who claims he was fraudulently imposed

8. *Technics of courts in dealing with allegedly excessive awards for traumatic neurosis.*

If a verdict is excessive, the trial court may require the plaintiff to remit a specified amount of the award, on pain of granting the defendant's motion for a new trial. If an appeal court desires, it can require a still further remittitur on pain of reversing the judgment and remanding the cause for a new trial. A conventional test of excessiveness is whether the verdict is so large, in going beyond fair compensation, as to shock the court's conscience and to require an inference that the jury was motivated by passion and prejudice.[26]

In practice, the courts act upon a variety of considerations as proper grounds for holding large awards excessive:

(1) Neurotic symptoms may be caused or aggravated by environmental influences or the circumstances of litigation, but the defendant is not liable for a worsening of plaintiff's condition produced by such independent causes.

(2) A judgment in plaintiff's favor may be reversed because the verdict rests on the jury's unwarranted assumption that traumatic neurosis is a permanent disability.

(3) Judgment may be reversed because the verdict is obviously excessive, but expert evidence adduced on the trial is not adequate to enable the appeal court to say how much the award should be reduced by remittitur.

(4) A verdict may be excessive because of the jury's failure to give proper weight to evidence that plaintiff's symptoms were in part due to pre-existing impairment of his nervous system or bodily health.

(5) A verdict may be considered excessive because of strong evidence pointing to conscious malingering.

(6) A verdict insupportable on the evidence calls for reversal of judgment, as, for instance, where the stimulus was patently inadequate to cause neurosis in an average person, and testimony indicated that P was already subject to that complaint.

Comparative competency of opposing experts may be of controlling importance in deciding inadequacy. Thus, a verdict which rests on testimony of general practitioners, not founded on a reasonably systematic examination of the nervous system, may require to be reduced or set aside where op-

upon is permitted by some courts to show his depressed state of health at the time, as one circumstance, even though it does not establish mental incompetency. Thus it has been held that a personal injury plaintiff who seeks to set aside a release on the ground of fraudulent procurement, is entitled to show that she was a profound neurasthenic at the time it was executed. Wilson v. San Francisco-Oakland Terminal Rys., 48 Cal. App. 343, 191 Pac. 975 (1920). As neurosis does not impair the intellect, such evidence should not be regarded as a ground for cancellation of the release, but merely as a circumstance directing closer scrutiny of the alleged fraud or imposition.

Early stages of schizophrenia, one type of psychosis, may produce symptoms similar to neurosis, causing an error in diagnosis, but any such confusion will be resolved as the psychosis progresses, and there is no evidence that neurosis progresses into schizophrenia.

[26] Carton v. Eyres & S. Drayage Co., 117 Wash. 536, 201 Pac. 737 (1921).

posite opinion evidence is given by defendant's experts, skilled neurologists, based upon exhaustive neurological examinations.

(7) If a plaintiff unreasonably neglects to minimize his own damages, this is adequate ground to refuse him full compensation for aggravation or prolongation of his injury.

If the prospective defendant tenders medical care which would have cured or ameliorated plaintiff's disability, and the latter rejects it, the principle mentioned applies. However, sending a case of traumatic neurosis into an ordinary hospital frequently makes symptoms worse. To make certain that his tender of treatment is adequate, the defendant should offer care in a quiet and proper place by a qualified neuropsychiatrist.

(8) Instances exist where appeal courts have reduced verdicts on the ground that plaintiff's lawyer made improper argument to the jury. As it is difficult to say how much the inflammatory remarks swelled the verdict, it is usually more satisfactory to deal with this prejudicial error by reversing the judgment and ordering a new trial.

Our opinion is that all cases of traumatic neurosis, and particularly those following trivial stimuli, should be compensated on a conservative basis with damages restricted to modest levels. This point of view seems to us to be required by several considerations, namely:

a. The basis of compensation depends upon subjective symptoms;

b. There are independent causes, of substantial weight, operative in all traumatic neurosis cases;

c. The defendant's act is usually a trivial stimulus which merely calls forth expression of a preëxisting neurotic diathesis or constitution, and thus tends to be a trigger mechanism rather than a substantial cause;

d. Diagnosis and evaluation depend on statements of the patient as to nature and severity of his symptoms, and there may be no adequate verification or method of objective measurement;

e. Malingering is very difficult to prove, but is often present, and almost always there is unconscious exaggeration of symptoms.

B. *Traumatic Neurosis in the Field of Insurance Law.*

Workmen's Compensation Insurance: The duty to insure the employed workman is statutory and the idiosyncratic person is not excluded.[27] He is

[27] Crowley's Case, 223 Mass. 288, 111 N.E. 786 (1916), allowed full compensation for disability due to aggravation by accidental injury of preëxisting dormant syphilis. Braley J. spoke the majority view as well as the rule for Massachusetts when he said in his opinion:

"The statute prescribes no standard of fitness to which the employee must conform, and compensation is not based on any implied warranty of perfect health or of immunity from latent and unknown tendencies to disease which may develop into positive ailments if incited to activity through any cause originating in the performance of the work for which he is hired. What the legislature might have said is one thing; what it has said is quite another thing; and in the application of the statute the cause of partial or total incapacity may spring from and be attributable to the injury just as much where undeveloped and dangerous physical conditions are set in motion producing such result, as where it follows directly from dislocations or dismemberments or from internal organic changes capable of being exactly located."

brought within the policy by being put to work, and is entitled to *some* compensation if his injury resulted from an accident sustained in the course and scope of employment.[27] Observe that we do not specify *how much* the claimant should recover, for this must be determined by deciding what part of the total injury is attributable to the accident.

War Risk Insurance: Here the government, as insurer, comes under a *duty* to pay benefits to the insured person in certain contingencies. There is no dereliction or breach of duty unless the insurer refuses to pay when a stipulated contingency has occurred, namely, proof by the veteran that he incurred a service-connected total and permanent disability before the date his policy lapsed for non-payment of premiums.[28]

Life, Health and Accident Insurance: The insurer, by contract, comes under a *duty* to pay benefits to the insured person if certain contingencies occur, such as disability or death, and these are not brought about by one of the excepted causes. Many such policies provide that they are void for breach of warranty if the insured takes out the policy without disclosing presence of a material disease. Accident policies often endeavor by their language to exclude liability for injury due in whole or in part to preëxisting disease. In practice these clauses are construed favorably to the insured.[29]

The reader will appreciate the fact that transactions which give rise to tort actions usually involve strangers, or the actor has not had occasion to examine the person acted upon. Very often the actor derives no benefit from presence of the person acted upon, and duty must depend upon risk of injury. That reasonable enterprise may not be discouraged, we argued that the law should hold an actor owes no special duty of care to the idiosyncratic person unless he knows or should know of the latter's excessive vulnerability.

Relations of insured and insurer do not involve two strangers. There

[28] It is not enough for P to prove that he was *permanently disabled* at the time the policy lapsed; he must also prove that he was then *totally disabled*. P does not establish his right to benefits if a partial disability at date of lapse did not become total until some subsequent time. (Attention is drawn to the fact that war risk policies are no longer being issued; in the present war the National Life Insurance available to service men insures against the risk of death only.)

[29] If a policy provides for payment of benefits in event insured becomes disabled, this includes functional as well as organic disease and thus traumatic neurosis or hysteria. Butler v. Prudential Ins. Co. of America, 117 Pa. Super. Ct. 367, 177 Atl. 335 (1935). But in view of the fact that traumatic neurosis is not entitled to be rated as a total and permanent disability, it is not clear how a claimant can ever establish his right to recover benefits under a clause (as in the Butler case) which requires such proof. In the Butler case there was some evidence that the neurotic symptoms were due to actual brain injury, and such a case may involve permanent injury.

If the disability clause requires not only "incapacity to transact any and every kind of business" but entire and continuous confinement to bed under a physician's care, a *neurasthenic* who cannot attend to business but is able to travel for his health, is not entitled to benefits. Bradshaw v. American Benevolent Ass'n, 112 Mo. App. 435, 87 S.W. 46 (1905).

Continuous progression of traumatic neurosis symptoms for almost two years after injury to eye, producing delayed disability, will not defeat right to benefits. Thompson v. Aetna Life Ins. Co. of Hartford, 177 S.C. 120, 180 S.E. 880 (1935).

Whether treatment for neurosis within two years prior to date of policy was treatment of a "serious disease, injury, or physical or mental condition" which would avoid policy was a jury issue in view of medical testimony. Potter v. Metropolitan Life Ins. Co. (Superior Ct. Pa.), 27 Atl. (2d) 703 (1942).

is a continuing relationship, ushered in by a contractual assumption of risk. Furthermore, the employer, the government, the insurance company not only can but do subject the inductee to rigid physical examination. They know or should know if the inductee is idiosyncratic. For all these reasons the *duty* assumed includes these frail fellows. We clinch our argument here by asserting that in the great majority of cases the inadequate personality can be detected by proper neuropsychiatric examination. The incipient or early neurotic can be spotted.[30]

Traumatic Neurosis in Workmen's Compensation Cases.

The Workmen's Compensation Law introduces a somewhat different philosophy regarding right to compensation for injury. We cannot ignore this fact in projecting a rationale for compensation of the traumatic neuroses.

At common law, the employee who sought to hold his master in damages for personal injury received in course of his work was required to prove some negligence or fault on the part of the employer. He was barred by his own contributory negligence or if it appeared that his injury was caused by the negligence of a fellow employee. He assumed the risk of injury from apparent hazards arising from working conditions or from the state of his master's premises. This is still true of an employer of one employee or of a number less than the statute provides for; moreover, the employee may elect his common law rights when he is employed.

Workmen's compensation laws have wrought an innovation: under them benefits are payable to an insured workman if he is disabled by an accidental

[30] The detection of such persons to the end of excluding them from the armed services has been a noteworthy social contribution of medical examiners.

Data re 21–36 year old registrants: Of two million registrants, it was estimated by the writers on the basis of 19,923 actual examinations by local boards and by Army induction stations, and on the basis of summary reports from local boards, that about 50 per cent were established to be unqualified for general military service, 900,000 for lack of physical and mental qualifications and 100,000 for lack of educational qualifications. Of these 900,000, Selective Service, on the ground of mental and nervous defects, rated 8,000 registrants as qualified only for limited military service and 30,000 disqualified for any military service. The Army, on the same ground, rated 19,000 as unqualified for general military service, thus yielding a total of 57,000 registrants (or 6.3 per cent of the 900,000 unqualified persons) rejected for mental and nervous defects. Of every thousand men examined, 18.2 showed mental disorders and 22.8 showed nervous disorders, a total of 41 per thousand.

ROWNTREE, L. G., MCGILL, K. H., and FOLK, O. H.: Health of selective service registrants, Jr. Am. Med. Assoc., 1942, cxviii, 1223.

Data re 18 and 19 year old registrants: This study, based on a sample of 45,585 reports of physical examination and induction, covering December 1942, and January and February 1943, showed that of white youths called up for physical examination, 23.8 per cent were rejected either at local boards or at induction stations, whereas the rejection rate for negro youths was 45.5 per cent. Of every thousand men examined, 27.6 were rejected for mental disorders and 14.8 for nervous disorders, or a total of 42.4 per thousand. Of these 42.4, 15.2 per thousand were rejected for psychoneurotic disorders.

The authors hasten to explain that while the rejection rate for the younger age group is about the same as for the older registrants, some caution should be exercised in drawing conclusions from this for the reason that "a large proportion of physically fit youths were not liable for examination either (*a*) because of previous enlistment in the armed forces, (*b*) because of programs that postponed examination and induction until a course of training had been completed or (*c*) because of employment in war industry or agriculture."

ROWNTREE, L. G., MCGILL, K. H., and EDWARDS, T. I.: Causes of rejection and the incidence of defects among 18 and 19 year old Selective Service registrants, Jr. Am. Med. Assoc., 1943, cxxiii, 181.

injury arising out of and in the course and scope of employment. The injury is measured by comparing disability to work after the accident with disability before it. If a workman afflicted with preëxisting heart disease is able to get to his job and perform it, but as the result of an accidental injury which would do little or no harm to the average person becomes disabled, this whole disability is imputed to the accident and so compensated. In contrast with tort law, personal injury is not compensable, unless scheduled, or unless it produces some disability to work as before. This difference in the basis for calculating the measure of damages in workmen's compensation cases and in tort law is a significant one. In tort law it is material that the vase was cracked before the accident occurred; in compensation law it is of no moment that the vase was cracked before the accident if it would still hold water. Thus prior impairment or idiosyncrasy of the workman is immaterial in compensation law, if the *whole* disability to work is due to aggravation of preëxisting poor health by accidental injury.[31] Even so, workmen's compensation insurance is not social insurance. Should society some day provide for automatic compensation of its disabled members, preëxisting idiosyncrasy or vulnerability will no longer be of consequence. The only issue will be: Is there a genuine disability or is the citizen a lazy loafer who is malingering? We have not yet attained to that Utopia and in the meantime it is important to recognize that the concept of *fault* in altered form is still used as a device for making the particular employer bear risk of injury by paying premiums. The *fault* is not common law negligence, but involves a new principle of responsibility and a partial step toward social insurance—incriminating the business on the score of risks produced in its conduct; *ergo,* the injury is not compensable unless it is an injury arising out of and in the course and scope of employment. From this it follows that if disability is made up of two parts, one due to accidental injury, the other arising independently of it, the claimant is entitled to compensation only in respect to the former. If a weakened or diseased heart is injured by an accidental strain, the resulting disability follows directly, and usually without suspicion of new and independent causes. Not so in the case of the neurotic personality: part of the resulting disability is due to the effect of the accident on the set stage, but new and independent factors always operate to exaggerate the disability. The defect is one in actual causation, and justice can be done only by restricting benefits to that part of the disability due to the accident. Some of the compensation commissions have perceived this logical barrier to full compensation of traumatic neurosis. If they find a claimant is totally disabled from neurosis, but believe the accident is responsible for only 20 per cent of the symptoms, they enter an award for a 20 per cent total disability. This device, one should note, may involve partitioning of causation, a scientific procedure foreign to the common law, which follows an

[31] The most spectacular instance is the practice, in a majority of jurisdictions, of allowing full awards for disability due to aggravation of preëxisting heart disease by accidental injury arising out of and in the course of employment.

"all or none" theory of causation. At common law, a defendant's conduct is either a substantial cause (sole or concurring) of the plaintiff's injury, or is not the cause at all. Apportionment of disability from neurosis between accident and independent causes is desirable to the fullest extent that the law of the jurisdiction permits. In jurisdictions where complete apportionment might be sanctioned, the medical examiner should endeavor to partition causation, and thus disability, between the accident and independent factors, as follows:

1. Percentage of disability from traumatic neurosis attributable to the accident (10 per cent to 50 per cent); the stimulus involved in the accident will be found to range from a mere trigger mechanism to a substantial cause apt to produce some nervous shock or symptoms in an average person.
2. Percentage of disability from traumatic neurosis attributable to pre-traumatic neurotic constitution or to non-compensable independent causes. (Deduct percentage determined above from 100 and verify by thorough study of pre-traumatic neurotic constitution, and of the post-accident factors likely to be aggravating or exaggerating symptoms.)

Obviously no exact mathematical formulae can be applied, for the apportionment between accident, pre-traumatic neurotic constitution, and post-accident causes requires sound clinical judgment of an impartial, competent neuropsychiatrist.

The pre-traumatic personality is investigated by appropriate inquiries directed toward determining what neurotic tendencies or symptoms were already present before the alleged injury occurred. These include:

1. Medical history: What doctors has patient seen and for what complaints? The medical history of a neurotic shows vague symptoms, oftentimes multiple, which do not incriminate any particular organ.
2. Past school record.
3. Matrimonial harmony or discord.
4. Attitude toward job. Nature of past employment. Work record and promotions. Ability to hold strenuous job. Neurotics tend to remain for long periods at jobs where special concessions of various kinds are made to them.

Some compensation acts would seem to authorize commissioners to exclude compensation both for preëxisting disease or neurotic diathesis and for disabling symptoms arising from subsequent and independent causes. What can be done should be done in both directions. It is likely that the present practice of granting full compensation for the aggravation of preëxisting disease by accidental injury is logically erroneous and socially unwise in that it tends to exclude those with disabilities from employment. It is true one can plausibly argue that as the employer, by medical examinations, has a sieve for sifting, he must assume the full risk of total disabilities partially due to accident and partially due to preëxisting disease or extra vulnerability.

Let us see how this doctrine works out in point of social effect. The average incipient neurotic is capable of holding a job and supporting himself. The employer knows, however, that a "tiny touch" may cause the incipient neurotic to develop a compensable total and permanent disability. Under the "full liability" theory subnormal or handicapped people will be weeded out, and excluded from industry. This is not theory but fact, well illustrated by the almost complete exclusion of epileptics from industry. If the doctrine for which we contend be followed, a medical auditor, referee or impartial examiner would carefully study the case and apportion the disability between accident and preëxisting causes. The insurer, and indirectly the employer, would not pay 100 per cent on a traumatic neurosis case, but only 10, 20, or 30 per cent, or what was fair in light of all facts. This policy would be eminently fairer to the employer and it would tend to make the impaired person employable on a just basis. We favor legislative changes in compensation laws designed to permit scientific apportionment of causation in all cases. The alternative is to see the practice grow of sagacious employers using medical examination to screen out of useful service those whose health may be slightly impaired but who are yet capable of a productive rôle in the industrial machine. The third alternative is a statutory provision authorizing the impaired employee to sign a disability "waiver" in going to work.[32] Such a waiver releases claims for benefits in respect to accidental injury sustained only because of a specified preëxisting defect. Statutory waivers help to make handicapped persons employable, but partitioning causation is a more scientific and equitable method of attaining the same objective, for it permits partial compensation.

Most workmen who develop traumatic neurosis can be got back to work. The process requires sympathy and patience on the part of employer and proper psychiatric care. After allowing compensation for traumatic neurosis as a matter of course, the judge may be surprised to find months or years later that the workman's condition is no better. At this point the court will feel constrained to cut off further compensation, and often this is done on the ground that the claimant has stayed away from work an unreasonable time or is malingering. This is a convenient fiction or reflects ignorance of the

[32] The Massachusetts Workman's Compensation Act, sec. 46, provides:

"No agreement by any employe to waive his rights to compensation shall be valid, but an employe who is for any reason peculiarly susceptible to injury or who is peculiarly likely to become permanently or totally incapacitated by an injury may, at the discretion of the department and with its written approval within one month of the beginning of his employment, waive his rights to compensation under sections 34, 35, and 36, or any of them."

This provision is expected to make many partially disabled war veterans employable. The Supreme Judicial Court of Massachusetts has not yet had occasion to define the phrases "peculiarly susceptible" and "who is peculiarly likely." However, Mrs. Emma S. Tousant, chairman of the Massachusetts Industrial Accident board, explains the waiver provision thus: Assume: . . . "a veteran had a bullet hole in his wrist. A bone had been removed from it, leaving his wrist weak. Now, if he sustained an injury at work which stemmed out of that wrist weakness, he could *not* get compensation. But if an elevator dropped and he broke a leg, he certainly would collect, just as any other workman in that elevator would collect, because that injury had nothing to do with the weak wrist." Boston Traveler, Tuesday, Oct. 5, 1943.

basic situation. Remember that benefits paid to a disabled workman are not so large as the earnings he is losing. He does not profit financially by continuance of his neurosis. He has initial symptoms which produce some disability. If he tries to return to work, a foreman not imbued with the ideal of gradual and sympathetic rehabilitation, may deepen the neurosis by putting him to work at once on his old job. The demands are too great, and the workman's failure aggravates his neurosis. During this time, the insurer's agents are apt to be investigating and questioning the reality of the claimant's illness. The claimant's financial obligations remain fixed while his income is cut in half. He may acquire feelings that he is the victim of social injustice. These factors cause the gorge of the workman to rise, producing psychological tensions which increase the neurosis still further. Some of our large industrial companies have practically no traumatic neurosis among their employees because they follow a program of sympathetic rehabilitation. The sick man is continued at his regular salary; the reality of his complaint is not put in issue unless frank malingering is involved, and the employee is given the best medical care. He is returned to his job at an early date under sympathetic oversight and by easy stages calculated to rebuild his lost confidence. It will be observed that everything possible is done to dissipate the sense of insecurity and frustration. The same rationale of rehabilitation will be required to restore veterans with war neuroses. Both in the compensation cases and in the veteran cases, a single lump sum payment is preferable to protracted payment of benefits. This should be coupled with provision for giving the neurotic a sense of security and a program of gradual rehabilitation, until his restoration is accomplished.

It is a common mistake in compensation proceedings, as in the tort cases, to regard traumatic neurosis as a permanent disability. It is not a provident thing to keep reopening awards and perpetuating installment payments, as some commissions do. Traumatic neurosis cases should be disposed of once and for all, by one award and not piecemeal.

Alleged Conversion of "Traumatic Neurosis" to "Compensation Neurosis."

Example: C developed a traumatic neurosis from an accident received in course of employment. This expressed itself as hysterical paralysis with certain side symptoms. C was hospitalized and under psychotherapy lost practically all his symptoms, so reporting to I, the commission's impartial medical examiner who had first diagnosed hysterical paralysis. In a few days the hysterical symptoms returned. I testified before the commission on application for further benefits, that C had been cured of hysterical paralysis due to traumatic neurosis and that return of symptoms was due to compensation neurosis. C's claim was denied on the ground that the disability from which he now suffered was not caused by the accident.

We do not recognize "compensation neurosis" as a condition apart from the original "traumatic neurosis," for the former is only one part of the total neurotic reaction to the original injury. The idea of a conversion in midstream from compensable to non-compensable neurosis is a tempting device

to enable curtailment of benefits in select cases, but it is not medically sound. Furthermore, we would stress the fact that temporary remission of symptoms in traumatic neurosis under special psychotherapy is no proof of cure. In such cases the neurotic symptoms are notoriously apt to recur.

On the other hand, as we have pointed out heretofore, a vulnerable person may begin brooding a long while after the accident about what might have happened to him and to those financially dependent upon him, thereby developing a late anxiety neurosis which should not be compensable. As pointed out in discussing the tort cases, proof of causal connection becomes more conjectural as the symptom-free time interval between alleged stimulus and effect lengthens. It may be denied if satisfactory "bridging symptoms" do not appear at once, or within a few hours or days, such symptoms being speedy and persistent complaints of pain, substantial nervous shock, or prompt and continuing deviations in psychologic behavior. The later a neurosis is in attaining "full bloom," the more reason there is to suspect that independent and non-compensable factors may be operative.

Traumatic Neurosis in War Risk Insurance Cases.

The government is employer of the soldier and the latter is truly a servant of the state, drawing modest compensation for the heavy risks to which he is exposed. We draw much nearer to the concept of social insurance when we encounter the war risk insurance policy. Courts should and do follow the principle of liberal construction in dealing with the legal claims presented by the veteran.

Thus, it is held that if the disability is a mental or functional condition, late diagnosis some months or years after lapse of the policy is not necessarily incompatible with the veteran's contention that he was totally and permanently disabled from a service-connected cause when the policy lapsed. This is put on the ground that such conditions are more difficult to discover and diagnose than physical injuries or organic diseases. It is further held that a rather long work record between lapse of policy and filing of claim does not disprove the veteran's total and permanent disability during the interim period, particularly if the evidence shows much shifting of jobs and unsuccessful efforts at adaptation to steady employment. He is not confined to evidence of his physical condition at time the policy lapsed, though this is the real issue, but is permitted to show the subsequent unfoldment of his disability in proving that he was totally and permanently disabled when the policy lapsed. These concessions are more safely made in respect to the psychoses than in regard to the neuroses.

War risk insurance issued during the first world war paid benefits for total and permanent disability incurred before the policy lapsed. Veterans who let their policies lapse upon leaving service, or shortly afterwards, have continued to file suits claiming a present disability had become total and permanent before the policy lapsed. In testing the alleged cause-effect relationship in a neurosis case, we cannot accept the hypothesis of a service-

connected causation where there has been a substantially symptom-free period of as much as four to six months after lapse of the policy before appearance of the characteristic symptom-complex.

C. *Recurrent Problems of Trial Practice.*

Rules of pleading in legal proceedings are intended to give each party notice of contentions his adversary will seek to prove at time of trial, thus eliminating surprise and affording him fair opportunity to organize counter-testimony. The pleader is required to set forth the ultimate facts upon which he rests his cause of action or defense, but not to plead his detailed evidence. In personal injury litigation, it is generally enough for a plaintiff to plead the medical diagnosis of the condition allegedly caused by a defendant's dereliction, this entitling him to prove, without pleading, all the symptoms characteristic of that medical complaint. This rule has been held applicable to traumatic neurosis. There is some question about the wisdom of so holding, for we have seen that traumatic neuroses are not ultimate entities of fixed connotation, but symptom-complexes. The symptoms form the basis of compensation, and they vary widely in their nature. For that reason a defendant is not given fair notice and opportunity to organize counter-evidence effectually unless the plaintiff be required to plead the type of the neurosis and its leading symptoms.

In some instances defendants have requested the trial court to charge the jury that traumatic neuroses, because of their subjective nature, require more positive proof or more conservative compensation. The usual response is for the court to decline, on the ground that such a charge would unfairly prejudice the plaintiff's case. The assumption underlying the requested charge is correct, for the several reasons we have discussed. The desired effect may be obtained by charges which stress more fully than heretofore the requirements of causation, and the duty to exclude from compensation symptoms produced or aggravated by extrinsic causes. Trial courts should also prevent, as far as possible, undue dramatization in the trial of traumatic neurosis claims. Such presentations tend to produce excessive verdicts, yet rules of court can hardly succeed in curtailing the dramatic possibilities entailed in trial before a jury. This being true, the need is all the greater for trial and appellate courts to scan awards with watchful eye and to apply freely the secondary corrective of reducing excessive verdicts.[33]

[33] Little can be done, for instance, under rules of court to curb melodramatic exhibitionism.

In Kress v. Sharp, 156 Miss. 693, 126 So. 650 (1930), a case of traumatic hysteria, plaintiff's attorneys put on a royal circus. One act was to have P examined in a room of the court house during progress of the trial, with several young ladies (X, Y and Z) present. P's condition was aggravated by this procedure and she went into hysterical exhibitions. X, Y, and Z were immediately put on the witness stand and conveyed their impressions to the jury, testifying, in effect, that P was a physical and mental wreck. The jury returned a verdict in P's favor for $25,000. The Mississippi Supreme Court took P's attorneys and physicians to task in a trenchant opinion. It held the verdict was grossly excessive and reversed the judgment for new trial on the limited issue of damages.

Malingering.

A recurrent problem is how to prove suspected conscious malingering. This issue may arise in any litigated case of neurosis, directly or inferentially.[34] Malingering cannot be predicated on momentary disappearance of a hysterical contracture when the patient's thoughts are diverted, as this is a normal phenomenon. Difficulty in proving malingering is increased by the fact that no two symptoms, however diverse, are incompatible in traumatic neurosis, for aches and pains from head to toe with multiple side symptoms, none pointing to a specific disease, are diagnostic evidence of psychoneurosis.

How, then, is one to test malingering?

1. A careful study of the pre-traumatic personality and history is valuable. If the person was an incipient neurotic one will find that he has been going to doctors with vague and diffuse complaints, that he has had difficulty in personal adjustments or relations, or that he works where the employer will make special concessions to his state of health. If this type of person is revealed, his neurosis is most likely real and any intentional malingering will be by way of exaggerating severity of his symptoms. The careful medical examiner can usually judge the extent of "staged" exaggeration.

2. If the person has no such past history, he may be a frank malingerer and one must focus sharply on adequacy of the described stimulus to produce traumatic neurosis in a person of average constitution.

3. Conscious malingering in respect to hysterical blindness or some other ailments, can be tested by trick mirrors or lenses and other apparatus designed to make the person believe one member of his body is being tested when in fact another is being examined.

4. Hysterical contracture, if it has not continued so long as to produce actual physical impairment, will disappear completely under anesthesia, and in this way can be distinguished from an organic lesion. This test demonstrates whether the contracture is organic or psychological in origin but proof that it is of the latter variety does not prove conscious malingering.

5. All sorts of methods must be used to prove that the subject actually performs the functions which he asserts have been lost. Claim agents may take motion picture "action shots," persons who call may find the hopeless cripple walking, or the like, and other evidence contradictory of the plaintiff's claims may be discovered. Where a plaintiff confesses in court that she purposely exaggerated the severity of her symptoms, this circumstance will require closest scrutiny of the amount of damages awarded by the jury, but will not upset it, if without reliance on plaintiff's testimony, one can see from independent medical evidence that the plaintiff did suffer substantial injury for which the award is not excessive.

[34] *Conscious malingering:* We adopt Wechsler's definition that "the individual becomes a malingerer only when he consciously and purposely, in order to deceive, to evade responsibility, or to derive gain, feigns illness and voluntarily tries to reproduce signs and symptoms which he really does not have, or extravagantly exaggerates minor ones which he has."

6. A defendant is entitled to have medical experts who have observed the plaintiff's demeanor and reactions on trial of the case take the stand and give any opinion they may have reached concerning presence of conscious malingering. One court has held that an expert cannot describe the plaintiff's demeanor, as the jurors can see that for themselves, but this seems to be an artificial and undesirable restraint, for the trained eye of the medical expert will see significant features which the layman might miss. Furthermore, an expert should always be encouraged to expose the basic facts upon which he rests his opinion.

The reader will see that traumatic neurosis is the flowering stalk of a previously planted seed, the recurrent breaking open of an old scar, the further damaging of a cracked vase so that it will not hold water until mended once again, indeed a veritable Pandora's box full of idiosyncrasies, multiple mechanisms and ambiguous causations. It has taxed the scientific technics of medicine, and we do not wonder that it should have strained the methods of the law now available for fixing a fair compensation.

We said in the beginning that the law-medicine problems which revolve around the traumatic neuroses are vexed and vexatious. We say so again. If we have put forward a useful rationale for orientating the problem of proof, we have gained our goal.

THE DIETARY FACTOR IN THE ETIOLOGY OF PERNICIOUS ANEMIA *

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ADDISONIAN pernicious anemia is known to develop in accordance with genetic, racial, climatic and geographic determinants. The hypothesis that the disease is of dietary origin has been advanced. If dietary deficiencies cause Addisonian pernicious anemia, they must explain the natural distribution and the natural history of the typical form of the disease.

The Natural Distribution of the Disease. Pernicious anemia is geographically distributed. It is rare in Asia, common in Northern Europe, Canada and the Northern United States. It is less common in the Mediterranean and Southern European areas, in South America and Southern United States.

Racially, it is extremely rare in the Asiatics, Negroes and Egyptians. Constitutionally, the light complexioned, fair haired types are susceptible; the pigmented types resistant.[1] There is a tendency to familial grouping in the susceptible races.

Climatically, in the United States, it varies in incidence according to Petersen and Mills,[2a] with the storm track areas. Smith [2b] ascribes the distribution to variation in effective solar radiation.

What Are the Essential Objective Criteria in Typical Addisonian Pernicious Anemia? Macrocytic hyperchromic anemia, glossitis, bone marrow and nerve tissue changes are not specific objective criteria. They may be produced by other mechanisms than that producing Addisonian pernicious anemia.[3] They are pathognomonic only when they have been proved to result from a deficiency of the special anti-pernicious anemia liver principle. The essential objective changes in pernicious anemia are those involved in the depletion of this essential liver principle. There are three such constantly associated objective findings in the patient in relapse, permanent histamine refractory anacidity, permanent reduction of Castle's intrinsic factor, and reduction of the stored anti-pernicious anemia liver principle. This appears to represent a primary composite triad of objective criteria, essential in the pathogenesis of the disease. The anemia, bone marrow changes, and nerve changes are secondary developments. Only as composite findings are the three criteria significant. The loss of the intrinsic factor of Castle, although essential, is not, alone, pathognomonic of Addisonian pernicious anemia.

It has been found to be absent in patients who had malignant destruction of the pylorus,[4, 5] in sprue,[6] and in multiple intestinal anastomoses.[7, 8] Castle

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observed the disappearance and return of the intrinsic factor in a patient who had intestinal stenosis.[9] It has been shown to disappear transiently during pregnancy.[10] The manner of its loss, not the loss per se, is its pathognomonic feature in Addisonian pernicious anemia.

Even the loss of the anti-anemic liver principle does not, alone, denote the disease. It can be induced by other mechanisms than that producing Addisonian pernicious anemia. It has been shown to occur with cancer of the pylorus [11] and in sprue.[6]

The mere existence of the triad is not pathognomonic of Addisonian pernicious anemia. It is essential, but it can exist theoretically in other conditions.[6]

The specific features about the triad are its specific chronology of development and the precise relationship of the findings to the disease. The anacidity is invariably present; it is a long standing precursor and is permanently refractory to histamine. The loss * of intrinsic factor is invariably demonstrable, and is permanent. The triad is constantly associated with no other disease. Although its components may theoretically occur in isolated cases of other diseases, they do not show the above precise characteristics.

The Natural History of Pernicious Anemia. The natural history of the development of this essential objective triad suggests a long period of evolution. The chronology is apparently a development in orderly sequence of anacidity, loss of intrinsic factor, and loss of the anti-pernicious anemia liver principle.

The period of anacidity alone represents the potential stage of pernicious anemia, the hazard varying from a negligible one in ordinary individuals [12] to a fairly high one in blood relatives.[13] The time of onset is variable, supposedly congenital in some, but in others it is known to be acquired.[13]

The potential disease enters the latent stage with the onset of reduction of the intrinsic factor. At this period, the fundamental etiologic objective change of the typical disease has occurred. There now exists the defective physiology producing reduction of the specific liver principle.

The active stage, marked by abnormal blood, bone marrow and nerve tissue changes, occurs when the stored anti-pernicious anemia liver principle drops below the critical level necessary for normal hematopoiesis and nerve nutrition.

The successive stages, potential, latent and active, merge into each other without sharp definition, marked by no significant transitional signs or symptoms. The gradual objective changes are apparently reflected in the insidious subjective changes described by Addison. "The patient can hardly fix a date to his earliest feeling of that languor which is shortly to become so extreme." This, then, is the natural history of typical Addisonian pernicious anemia. Definitive data as to the etiologic effect of diet must ade-

* By the term "loss" is meant that there is insufficient intrinsic factor to produce a significant reticulocyte response by a biologic assay. Goldhamer has shown that a reduction, not a "loss," actually occurs.

quately explain this natural history of development. Anemias which are not produced by this orderly mechanism are not typical pernicious anemia and can be excluded from this study.

The cause of the typical form must be determined before considering the atypical. Anemias without achlorhydria are atypical. Anemias associated with retention of intrinsic factor are not Addisonian pernicious anemia. Anemias in patients who have lost intrinsic factor but not the hydrochloric acid secretion are not typical.

Can Diet Deficiencies Produce Either Typical Addisonian Pernicious Anemia or the Early Essential Objective Changes in Proper Sequence? We may approach the problem by asking the following questions:

1. Is the incidence of the disease higher in areas where undernutrition is prevalent?
2. Are the diets prior to the onset of Addisonian pernicious anemia deficient?
3. Do individuals with known, long standing diet deficiencies develop this form of anemia or its essential early criteria?
4. Can dietary deficiencies produce the essential findings in experimental animals?

Is the Incidence of Addisonian Pernicious Anemia Higher in Areas in Which Undernutrition Is Prominent? There is no correlation between the natural distribution of the disease and poor nutrition. It is found chiefly among those who use a diet high in protein and meat and are well fed. It is lowest among the people where famine, undernutrition and avitaminoses are common. In China, Yang and Keefer [14] found prior to 1931 only four of 25,000 hospital admissions who had pernicious anemia. In 1941, Snapper [15] said that in the admissions to the Peiping Union Medical College Hospital since 1921, only six patients could be found in whom the diagnosis seemed to be justified. In all six, however, some signs could be found which seemed to distinguish them from real pernicious anemia. He does not believe that they suffered from genuine pernicious anemia. In contrast, in North America three or four in every 1,000 patients admitted to general hospitals are found to have pernicious anemia.[16] In India, where Wills [17] described tropical macrocytic anemia which she attributed to deficient diets, pernicious anemia is practically unknown. There are few authenticated instances.[18] Erulkar [19] speaks of Addisonian pernicious anemia but described no patients. Tropical macrocytic anemia is also prevalent in the Malay States, China and the West Coast of Africa. In these areas, also, pernicious anemia is rare. In Java, all gradations of undernutrition and avitaminosis are always present. If loss of intrinsic factor and development of pernicious anemia were attributable to either acute or chronic undernutrition, it should be reflected in an increased incidence of the disease in Java. De Langen [20] states that during a period of 20 years he did not encounter one patient among the natives.

Are the Diets Prior to the Onset of Addisonian Pernicious Anemia Deficient? Few detailed studies of the diets preceding the manifestations of pernicious anemia have been made. Minot and Murphy, in their classic paper in 1926, said: "We have noted it is not uncommon for these patients to have consumed throughout life unusually large amounts of food rich in fats. Patients with pernicious anemia also may give a history of partaking for years of some other type of one-sided diet." Cornell [21] studied the diets used by 26 patients during 10 years before the onset of symptoms and signs of pernicious anemia. He concluded that "on the whole, these individuals have consumed fairly average diets. Their diets, in most cases, were the same over the ten year period as those of their nearest associates, who did not develop pernicious anemia. It is probable, therefore, that pernicious anemia is not due, fundamentally, either to excess or deficiency of any type of food."

Ungley and James [22] studied the diets used by 15 patients preceding the onset of pernicious anemia. Six had used a diet especially deficient in meat and green vegetables. The remaining nine had used diets that appeared to be normal. They found no difference in the preceding diets of those with and without spinal cord lesions.

Unfortunately, dietary histories of patients prior to relapse do not furnish precise data. It cannot be said whether any deficiencies noted preceded or followed reduction of the intrinsic factor. Thus, diet abnormalities found years before relapse still might be due to the anorexia and taste perversions of the early active stage. They might represent concomitants of the early stage and not precursors. The aversion to meat reported by many patients for a variable period is probably an abnormality of the latent and early active stage. There are apparently no precise data upon which any marked diet deficiencies in individuals with Addisonian pernicious anemia may be based, save for the period just before manifestation of the disease.

Can Dietary Deficiencies Produce Addisonian Pernicious Anemia? Specifically, can dietary deficiencies cause a loss of the antipernicious anemia liver principle by the mechanism by which it is lost in typical Addisonian pernicious anemia? The nature of the disease precludes any planned human experimentation with prolonged diet restriction. Natural experiments are available, however.

In India, many Mohammedan women live on diets largely vegetarian, high in carbohydrates and low in protein and the vitamin B complex. This produces, in many instances, a macrocytic hyperchromic anemia, but hydrochloric acid is usually retained in the stomach. The anemia does not respond to the Dakin-West anti-pernicious anemia fraction (Anahaemin)* [23] and, thus, is apparently not due to a deficiency of the specific antipernicious anemia principle. The dietary factor lacking cannot be Castle's extrinsic

*Anahaemin (British Drug Houses) is an extract prepared by the method of Dakin and West.

factor and the anemia cannot be ascribed to the mechanism which operates in Addisonian pernicious anemia.

In Northern China, Snapper [15] says the diet is generally deficient in protein, calcium, vitamins A, C and D but is sufficient in vitamin B. Here a macrocytic anemia very similar to that occurring in India results, so deficiency in vitamin B cannot be the factor in its production.

In Puerto Rico, diets deficient for years in meat, milk, eggs, whole grain cereals and butter are found prior to the development of sprue.[6] The majority of those with macrocytic anemia retained intrinsic factor sufficient to provoke a significant reticulocyte response after the administration of beef muscle. Loss of Castle's intrinsic factor was demonstrated in two instances by a biologic assay. In one, loss of the anti-anemic liver principle occurred. Hydrochloric acid was retained in the stomach. In the majority of these patients with sprue, there was retention of acid and of intrinsic factor. There was no constant relationship of the essential triad as is found in pernicious anemia. Therefore, in sprue even though certain components of the triad occasionally may be present, they are seldom all present, the characteristic sequence of their development does not occur, and they bear no precise etiologic relationship to the anemia. The deficient diet associated with the production of sprue does not induce a macrocytic anemia which can be explained by the mechanism producing pernicious anemia.

In the Southern United States, diets deficient in calories, protein, calcium, phosphorus and the known vitamins over a period of years lead, not to the development of pernicious anemia, but to pellagra. Macrocytic anemias with loss of hydrochloric acid and Castle's intrinsic factor may occur, but it is not a constant association.[24, 25] The anti-pernicious anemia liver principle was not lost in the one case tested.[26] Achlorhydria occurs in about 70 per cent.

Response to the Administration of Liver Extract. The macrocytic anemias of the tropics, of pellagra, and of sprue respond to the injection of liver extract, and for this reason they have been ascribed to a deficiency of the anti-pernicious anemia principle. Cohn's liver fraction G, which is usually employed, contains two distinct anti-macrocytic anemia principles—one the specific-pernicious anemia principle, the other the anti-macrocytic anemia principle effective in tropical macrocytic anemia.[27] Response to the administration of liver extract by injection apparently cannot establish that an anemia is due to deficiency of the specific anti-pernicious anemia principle unless a more purified extract, such as the Dakin-West fraction, is employed.

Tropical macrocytic anemia does not respond to the Dakin-West (Anahaemin) fraction.[23] Wills described [23] one instance of macrocytic anemia of sprue which did not respond to the administration of the Dakin-West fraction. Such data are meagre, because in most instances treatment is with Cohn's fraction G. Other data suggest that the deficiency causing these anemias is not one of the specific anti-pernicious anemia principle. A much

larger dose of liver extract is needed for nutritional macrocytic anemia, and for the macrocytic anemias of pellagra and sprue than is needed for pernicious anemia in relapse.[28]

Clinically, these macrocytic anemias have more similarities to each other than they have to Addisonian pernicious anemia. They do not show the increased hemolysis of pernicious anemia, nor the frequency of neurologic involvement. Diet deficiencies are common to all, and the character of the deficient diets is similar.

To summarize, deficient diets in man over a period of years may be demonstrated to produce macrocytic anemias, but not commonly by the mechanism operative in Addisonian pernicious anemia. They are apparently not due to deficiency of Castle's extrinsic factor, to loss of the intrinsic factor nor to loss of the specific anti-pernicious anemia principle save in such rare instances that it cannot be of etiologic significance.

Diet deficiencies apparently do not produce either manifest pernicious anemia or the early essential objective changes.

Can Dietary Deficiencies Produce the Essential Findings of Pernicious Anemia in Experimental Animals? Dietary observations on experimental animals must face fundamental objections. Pernicious anemia does not occur spontaneously in animals.[29] Similar syndromes are analogues which serve only to direct the study of the human prototype. It is difficult by any means to produce a satisfactory picture of pernicious anemia in an animal which might be used as a test animal for the determination of liver potency.[30] Few lower animals have the same mechanism of production of the anti-pernicious anemia liver substance as is found in man. Only in the hog does the physiology of production of this principle seem similar enough to justify its use as an experimental animal. This similarity is a broad one, however, and cannot be accepted with respect to details without inducing faulty inferences. This has been illustrated by the inference that Castle's intrinsic factor in man was produced in the pyloric glands and Brunner's glands because this was true in the hog. Fox [31] demonstrated by biologic assay that the intrinsic factor in man was probably secreted in the fundus type of gland and not in the "pyloric gland organ" as occurred in the hog.

Only in the hog has the essential triad of objective findings been produced by diet deficiencies.[32] This followed the use of a modified Goldberger-Wheeler diet. The same diet in man, however, does not produce Addisonian pernicious anemia, nor the essential triad. It usually produces pellagra.[33] The same diet in the dog produces black tongue, not the essential triad.

Deficient diets administered to monkeys produce conflicting results. The marked nutritional macrocytic anemia produced by Wills [34] was not analogous to pernicious anemia as it did not respond to refined liver extract effective in man. Bussabarger [35] could not produce a similar anemia in monkeys by a similar diet.

The application of such results obtained on experimental animals to man is confusing rather than illuminating. Diet deficiencies which produce the

essential objective triad of pernicious anemia in the remote species, the hog, fail to produce pernicious anemia either in the nearest phylogenetic species to man, the monkey, or man himself. Results in experimental animals can be of crucial significance only when diets which produce the triad in animals are used in man and produce pernicious anemia. Acceptable data must be based upon example rather than upon analogy.

Discussion

By correlation of diet with manifest Addisonian pernicious anemia and with the early essential objective criteria, no essential relation can be established between diet deficiency and the incidence of manifest or early Addisonian pernicious anemia. Diet, apparently, is not a factor in initiating the disease. Is the geographical distribution, then, dependent upon climatic or upon racial determinants? In the same climatic environment, there is definite racial difference in the incidence of Addisonian pernicious anemia. Friedlander[1] found this true in Boston. In Java, only the white races develop the disease. Pure blooded negroes do not develop pernicious anemia in the same climate as Caucasians.

Where the racial factor is constant, and the climatic factor is variable, varying results occur.

In China and Japan, although the climate varies radically in Northern and Southern areas, there is no varying effect upon the incidence. There is little or no pernicious anemia in any area in either China or Japan. Pure blooded negroes apparently do not develop pernicious anemia in either the Northern or Southern states in the United States. Among the white races of the United States, however, there is a lower incidence of the disease in those living in the Southern states.

Climate, apparently, is of secondary importance to race in producing the incidence of pernicious anemia. This is probably true, also, of Javanese, East Indians and other Asiatics. These races are relatively immune, regardless of the environment. In other races, exemplified by the white races in the United States, among whom the incidence is relatively high, climate is a factor.[2]

Racial factors best explain the national geographic distribution. Purely racial factors are hereditary factors. If the loss of intrinsic factor is a racial trait, it must be an hereditary trait. Heredity thus emerges as apparently the only adequate explanation for the natural distribution and the natural history of Addisonian pernicious anemia.

An inborn genetic defect is accepted as the cause of loss of intrinsic factor in many individual instances. The reluctance to accept this as the cause of all pernicious anemia has been due to an inability to demonstrate it as the cause in certain individual instances, and the uncertainty as to the parts played by race, diet, climate and geography.

Inability to demonstrate a family pedigree in an individual patient con-

stitutes no valid objection to the theory of hereditary transmission. A recessive gene carrying a genetic fault may pass undetected through an unlimited number of generations. Hereditary predisposition would appear to be responsible for the disease both in certain races and in certain individuals.

The following hypothesis for the genetic origin of Addisonian pernicious anemia seems compatible with the available data.

Faulty genes tending toward loss of Castle's intrinsic factor are apparently distributed in varying quantities in different races. The genotype in a rigorous climatic environment loses gradually the intrinsic factor but tends to retain it in warmer climates. Dietetic external conditions have no effect upon the rate of development of this loss. During the gradual depletion, however, a diet rich in extrinsic factor may postpone or a diet containing a frequent ration of liver may prevent the manifestations of the active stage of the disease.

Since liver is rarely a constant dietary ingredient, those individuals with the genotype for pernicious anemia eventually will manifest the disease if they live long enough. In the races where the genetic fault is comparatively common, the hereditary nature is evidenced by a frequent increased familial incidence, and a tendency toward a constitutional or hereditary type.

The acceptance of heredity as the cause of Addisonian pernicious anemia would make a program for the prevention of the disease feasible. The potential cases should be found among the blood relatives with achlorhydria.[13] Grouping of all blood relatives by the histamine gastric analysis into those with acid and those without acid would identify the vast majority of the genotypes who later will develop the disease.

Conclusions

1. Diet deficiencies cannot account satisfactorily for the production of Addisonian pernicious anemia.

2. The natural distribution, which is racial, geographic and climatic, can be adequately explained by hereditary factors.

3. An active program for prevention is thus justified by which potential cases may be sought among blood relatives.

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PSYCHOTHERAPY *

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PSYCHOTHERAPY may be defined as treatment by means which appeal to both intellectual and emotional functioning of the individual. It uses procedures which require special knowledge and training. In addition to, but also independently of such procedures, psychotherapy makes use of and is inadvertently influenced by factors involving the therapist, other persons in contact with the patient, and events affecting him. This definition implies that psychotherapeutic influences may be present in any medical treatment, in any relationship of the patient with others, in anything he is subjected to or comes in contact with, insofar as such varied relationships, things, and events somehow affect the patient. Such a broad definition necessarily postulates that aside from psychotherapy as practiced by specialists, psychotherapy is being carried out on a much larger scale by physicians practicing any branch of medicine, by psychologists, educators, social workers, nurses, and others coming in contact with the patient, although they may not intend to use and remain unaware of using psychotherapy.

Is Psychotherapy an Art or a Science? The answer is that it is both. And so it may be added, is any other medical treatment and the practice of medicine itself. The vast application of psychotherapeutic influences, whether used knowingly or not, and the varied success attained by the uninitiated account for the much credited contention that it is an art; further arguments in favor of its being an art point out that it deals with so-called "intangible elements" of the human organism. Of course, intellectual and emotional reactions of a person are different from physiological reactions, the latter being, so to speak, palpable physical phenomena. Psychological reactions are nonetheless observable and lend themselves to study just as comprehensively as many a physical or chemical phenomenon. Bear in mind that human beings form an opinion of the prevalent personality traits and modes of behavior of their friends. On the basis of their knowledge acquired from observation they have a pretty accurate idea of how those friends might react under certain known circumstances. It is obvious, however, that in the practice of psychotherapy, as in that of any other field of human endeavor and activity, aside from knowledge and experience, the personal equation of the therapist plays a rôle which cannot be too strongly emphasized. Not all those trained similarly in the same surgical clinic turn out to be surgeons of the same ability. So it is, generally speaking, with psychotherapists. Adequate or inadequate application of psychotherapy in individual cases will be determined not only by the extent of the experience

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and knowledge of the psychotherapist but also by his skill in making use of his experience and knowledge.

Scope of Psychotherapy. Although the aim of psychotherapy and its procedures is psychological in nature, it does not follow that so-called physical diseases, i.e., abnormal functions of organs, are outside its domain. Insofar as certain somatic disorders of functional origin are recognized to be of psychogenic origin, the rational therapy in such instances will be the one which aims primarily to treat the cause—the abnormal function of the person—rather than essentially the result, i.e., the abnormal function of organs. Moreover, even in certain of the so-called organic-structural diseases, psychotherapy may be a useful adjunct for the reason that disturbed function of organs affects more or less the intellectual and emotional functioning of the person—the host of the physical disease. Thus, the field of psychotherapy is not limited to psychiatry—to treatment of psychotic and psychoneurotic patients. It should be included in the therapeutic armamentarium of medicine—in the treatment primarily of physical diseases of psychological origin and also, within certain limits, of organic-structural diseases.

Problems. Two sets of problems face the psychotherapist: (*a*) Medical problems, and (*b*) problems deriving from the interrelationships between the patient and his immediate environment or community. Although it is not possible to draw a clear-cut demarcation between these two types of problems, nevertheless, they offer distinct points which need special emphasis.

1. The medical problems include complaints by the patient regarding his physical or mental health or both. In certain instances the sole or main complainer will be not the patient but those in contact with him, the emphasis being on the trouble caused by the patient's behavior to others in his environment. However, the outstanding common feature of the complaints either by the patient or by those in his environment, or by both, is that they are centered on the patient's health. Whatever opinion is prevalent as to the causal relationship between the ill health of the patient and his position in the environment, the fact remains that neither the patient nor his relatives and friends nor others in contact with him bring forth human relationships as a special problem.

2. The other problems with which the physician-psychotherapist must cope include human relationships as the central issue, though there may also be bodily complaints. The patient himself, although feeling healthy in every respect, complains of being unsuccessful in school or in his occupation, of being unpopular, of not being able to adjust adequately in his family and in his social relationships outside his home. He may or may not recognize his own faulty behavior; he may shift the blame wholly or in part to others, and in this he may be not wholly incorrect. In other instances the patient himself feels well and contented, but his behavior, attitudes, whether he recognizes it or not, make life uncomfortable for those around him. Those others, however, or some of them at least, may be not wholly correct in

blaming the patient entirely and not finding any fault whatsoever with themselves.

Thus the psychotherapist, whether he deals only with essentially medical problems or with problems apparently involving only social relationships, must bear in mind two possibilities: The one is that even though the patient's difficulties are wholly concentrated on his bodily feelings, his family and social relationships may also be potent factors. The other possibility is that in case the whole emphasis is laid on the patient's inadequate social relationships, or whenever the social relationships of the patient present a problem at all, one must not forget that in any relationship there are at least two partners. One of the prerequisites of adequate psychotherapy, therefore, is to bear in mind the possibility that under certain circumstances the treatment of the patient cannot be expected to be successful unless and until it is supplemented by changing his position in the environment or by trying to take care of the inadequacies in the environment.

Modes of Approach of Psychotherapy. The ultimate goal of psychotherapy is to attack the very cause of the patient's difficulties. It aims, therefore, to derive its methods from the etiologic and pathogenetic concepts prevalent in psychopathology. Like medical treatment, psychotherapy aims to be etiologic and pathogenetic or genetic-dynamic in the psychiatric parlance. However, etiologic and pathogenetic treatment, being necessarily restricted by limitations in our knowledge of etiology and pathogenesis, must yield to other less ambitious modes of treatment. For the sake of an orderly discussion, I suggest treating the varied psychotherapeutic procedures under two headings:

I. Non-Specific Psychotherapy
II. Specific or Genetic-Dynamic Psychotherapy

I. Non-Specific Psychotherapy. As the term suggests, this therapeutic approach aims to relieve the patient of his troubles, as far as possible, without claiming to attack specifically the roots of the evil. To use an analogy, this therapeutic approach is comparable to the so-called symptomatic treatment in medicine, although the guiding principles of the two therapies are totally different. Medical treatment aims to treat the abnormal function of organs, or to build up the physical resistance of the organism or to do both; psychotherapy directs its means of attack solely toward the mental functioning of the person, regardless of the apparent nature of the disorders —physical or mental. Non-specific psychotherapy uses various procedures, to all of which applies at least one common denominator, i.e., the absolute prerequisite of good rapport between the psychotherapist and the patient. In this patient-physician relationship the partners are not equals; it is a relationship of a weak person who seeks the help of a strong one. The patient believes in the superior knowledge and ability of the physician he chooses to help him. This faith creates a very fertile background for mental influences.

With this preparedness of the patient to receive those influences, the physician has the opportunity to exercise his ability to affect the intellectual and mental functioning of the patient. This is being accomplished through various ways and means, some of which operate even without the active participation of the psychotherapist. Thus the very fact that the patient is aware of the accessibility of the care he wants is apt to have a beneficial effect. Furthermore, any happening which will heighten the prestige of the therapist outside his actual contact with the patient will also strengthen the passive beneficial influence of the former on the latter. When the patient faces the psychotherapist, another also rather passive factor is therapeutically effective. I allude to the therapist's attentive and sympathetic listening to the patient's story of his difficulties. The benefit the patient gets from this phase of contact with the therapist may be attributed to several factors: The therapist's desire to understand the substance and nature of the patient's troubles and, above all, his recognition of the reality of these troubles, in contrast with the attitude of those who heretofore have shown the patient, explicitly or implicitly, unmistakable evidence of their feelings that his troubles were more imaginary than real. This display of respect, understanding, and a humane sympathetic attitude by the psychotherapist—"who knows better"—toward an individual who is burdened with feelings of inadequacy, who is a weakling insofar as alone he cannot cope with his difficulties, has tremendous therapeutic potentialities. In the process of recounting the story of his troubles the patient finds a certain relief from emotional tension. And last but not least,—still in the phase of passive psychotherapy—from these various factors, each of which has its relative therapeutic value, the patient derives hope, another very potent therapeutic agent.

This part of the psychotherapeutic contact, called, somewhat artificially and only for didactic purposes, the passive part of psychotherapy (attentive listening by the therapist to the patient's story and his attentive attitude toward the patient constitute action), is accompanied and followed by the therapist's more active participation. The tools used in the active phase of non-specific psychotherapy will be only mentioned here: Reassurance, persuasion, suggestion, and the extreme degree of suggestive action, hypnosis.[1, 2, 3] It should be pointed out, however, that hypnosis in certain of its aspects (as an adjunct therapeutic procedure) belongs rather to the specific (genetic-dynamic) psychotherapy.

To reëmphasize the essential factors entering into the non-specific psychotherapy, my description of the latter begins with the statement that procedures it uses require, first of all, good rapport between the psychotherapist and the patient. Two factors determine its establishment, the one being with the patient and the other with the therapist. The patient must willingly accept the treatment and be receptive of mental influences, reassurance, persuasion and suggestion. Suggestibility, an average normal human trait, becomes abnormal in both of its extremes—above and below the average.

For the purpose of non-specific psychotherapy, suggestibility above that of the average run-of-the-mill person is useful. Although it is a characteristic personality trait, it is important to bear in mind that it is apt to be strengthened or weakened by another factor which rests with the therapist. The latter must inspire in the patient, and also in others in contact with the patient, respect and authority. The higher the therapist's professional standing and his prestige in the community, the greater his chances to have an influence on the intellectual and emotional functioning of his patient. Moreover, he must be able to impress the patient as being not solely and not essentially interested in the "case" in a detached, scientific manner, but his attitude must convince the patient that his physician has the best human feelings for the patient's suffering and that his sole object is to help him. This combination of professional authority and sympathetic attitude on the part of the psychotherapist plus a patient who is a willing and capable recipient of mental influences constitute the ideal background for psychotherapy, more especially the so-called non-specific form. Unfortunately, like many other good things in life, these very ideal conditions for successful psychotherapy also contain the seeds for reverses, apt not only to jeopardize the reasonably expected good therapeutic results but also to create new troubles or to magnify the existing ones. I allude to what may be called "Negative Psychotherapy."

Negative Psychotherapy. Because of the confidence of the patient in the professional expertness of the physician, confidence strengthened by the affective component in their relationship, the physician must constantly be on his guard, i.e., he must always remember that whatever he does or says is apt to have an impact on the patient. One very serious difficulty in the therapist's position lies in the fact that, as in any other human relationship, his similar mode of behavior under similar circumstances may impress various patients and relatives in entirely different, albeit, opposite ways. Unfortunately it does happen now and then that the irreproachable, highly qualified behavior of the physician produces entirely unexpected adverse effects on the patient, either directly or through the criticism of the relatives and friends of the patient. A colleague of mine of a very high academic standing and equally high professional reputation, in the rôle of a consultant, took great care in carrying out a very comprehensive physical examination which consumed more time than an average ordinary physical examination does. The patient's immediate reaction was that the "professor" did not feel sure of himself; otherwise, it would not take him so long to carry out the examination. The effect of the physician's attitude on the patient, faultless and even praiseworthy as it may be, depends not on his attitude alone but also on the personality make-up of the patient and of those close to him. However, experience shows instances in which the physician's behavior is manifestly at fault and has definitely harmful effects on the patient. One speaks in such instances of "iatrogenic diseases," i.e., functional disorders of psychogenic

origin for which the physician is to be blamed.[4] By that one means that certain remarks made by the physician to highly suggestible individuals are responsible for causing or protracting complaints of various illnesses. Here the reader's attention is called to the following galaxy of physician's remarks to patients: "You should never go out on the street alone. You may collapse any minute." "Your arteries are slightly hardened." "Your aorta is a little enlarged." "Touch of TB." "Your heart is small." "Grumbling appendix." "Slightly raised blood pressure." "Flabby heart." "Instead of a heart you have a piece of fluttering cheese cloth." "The cells of your stomach are dead." "Neurotic," "psychoneurotic," "psychopathic," "neuropathic," "inferiority complex," "mother fixation." Whatever significance those terms might have had to the doctors who used them, they were accepted by those concerned as meaning disease. These allegorical and metaphorical expressions testifying to a sense of humor, imagination, and literary ability of their authors certainly were used under inappropriate circumstances and indeed had adverse effects on their patients.

II. *Specific—Genetic-Dynamic—Psychotherapy.* By definition this treatment is expected to derive from the knowledge of the "dynamics" or "mechanisms"; i.e., the knowledge of the cause or causes of the illness and their mode of action. This definition needs to be supplemented by the statement that it is not enough and not even the essential thing that the therapist alone feels that he knows the dynamics. It is, in addition, absolutely necessary that the patient himself accept intellectually and experience emotionally what appears to be the causative dynamic factors, that he, so to speak, relive the experiences which had become the genetic-dynamic factors of his troubles. The question arises, how does one go about it in eliciting the dynamic material? How does one proceed to make the patient face his past experiences, to make him grasp and feel their significance in his present troubles? The choice of the method will be determined by the therapist's concepts of psychopathology. The therapist who holds the view that only those experiences in the past life, and particularly the experiences of early childhood, which the patient apparently has completely forgotten, are the essential dynamic factors will regard Freud's psychoanalysis as offering the most significant psychopathological concepts and the free association technic as the only valid and thorough therapeutic method. On the other hand, the therapist who has the conviction that experiences occurring in any phase of the life of the patient may become potent dynamic factors in his illness will choose the method of studying the patient throughout his life. The study will deal with life experiences of which the patient is more or less completely aware and those of which he becomes aware in the process of discussion of events, experiences which may or may not have any apparent connection with the seemingly forgotten experiences. It will also deal with those emotionally colored thoughts, desires, strivings which may be only faintly recognized and not accepted by the patient and which sometimes become, so to speak,

revealed to him through his dreams. In short, the two types of the genetic-dynamic psychotherapies derive from the concept that the present difficulties of the patient are determined by his past life experiences. The very essential difference between them lies in the fact that one type relies essentially if not exclusively on experiences buried in the unconscious, and the other relies essentially on either spontaneously conscious life experiences or those which can be brought into consciousness through discussion. Thus, to reach their aim, the two types of therapeutic procedures have to analyze the mental functioning of the patient, i.e., their tool is analysis of the psyche. However, the term "psychoanalysis" is generally identified with a special technic —the so-called free association technic. The therapist who analyzes the mental functioning of the patient, in other words, who does psychoanalysis without using the special free association technic, has no trade name to offer which would adequately convey the very essence of his therapeutic procedure. The term "distributive analysis" [3] is certainly not telling. It would seem rational to apply the terms "psychoanalysis" and "psychoanalytic" treatment to any therapeutic procedure which tries to influence both the intellectual and emotional functioning of the patient through a study, *an analysis*, of his personality function, i.e., reactions to experiences throughout his life, experiences of which he is aware or unaware. One may further be more specific in making a distinction between: 1. Genetic-dynamic psychotherapy which makes use of the free association technic.[1,2] 2. Genetic-dynamic psychotherapy which makes use of the personality study, i.e., the study of the function of the person throughout his life under varied circumstances.[3, 5]

For current use the following terms are suggested:

1. Psychotherapy with the use of the free association technic.
2. Psychotherapy with the use of the personality study.

Psychotherapy Which Makes Use of the Personality Study. Only broad, general principles will be outlined here.

This type of psychotherapy finds a sound background in Meyer's teaching of psychobiology. Rennie in his recent formulation correctly called this type of treatment psychobiological therapy.[5] Its procedure consists essentially in discussing with the patient not only his complaints and problems for which he seeks the help of the therapist, but also varied problems and topics, some of which obviously are related to his difficulties, whereas others have no apparent connection with them. It should be noted that whatever the topics might be, their discussions have one common goal: to further the study by the patient himself of his modes of reactions to life experiences. To begin with, the discussion deals directly with and centers on the complaints and problems formulated by the patient himself and by those in close contact with him. In certain instances, the therapist will be faced chiefly with complaints by others rather than by the patient himself. On the other hand, the patient may be right in his conviction that the difficulty lies not with

him but rather with those who complain about him. Sometimes the environment, although in itself not unusual, is one that does not click with certain personality trends of the patient—his idiosyncrasies, susceptibilities, modes of acting—which again by themselves cannot be considered as being much beyond the normal range of human reactions. One of the tasks of the therapist may then be to deal with the environment, by changing its attitude toward the patient or by removing the patient from an environment unwholesome for him. I suggest that in so handling the situation, one accomplishes an important part of genetic-dynamic therapy, inasmuch as an attempt is made to bring about the patient's recovery or improvement by trying to affect the cause or causes of his difficulties or at least certain factors contributing to them. In most cases, however, whether or not the environment of the patient is to be blamed as an adverse contributing factor, there must be something wrong with the patient himself, since, although he cannot take it, cannot find a *modus vivendi* in his environment, yet he does not attempt to free himself from it, or is unable to do so.

The problems the psychotherapist has to cope with are not only those of maladjustment. The mutual relationship between the patient and his environment may not give rise to complaints from either side. The patient's behavior may be such that it does not in any way adversely affect his environment, nor is the latter objectionable to the patient. Nevertheless, while carrying out his daily activities adequately and behaving satisfactorily, the patient himself may be the sole sufferer, being badly in need of medical and psychiatric help. The necessary conclusion to be drawn from the statements made thus far is that it is the therapist's duty first and foremost to try to learn as much as possible about the complaints and problems involved as they concern both the patient and his environment; i.e., to obtain as comprehensive a history as possible, gathering information not only from the patient but also from whatever source information may be available.

Taking the History of the Illness and Psychotherapeutic Interviews. In the discussion of the non-specific psychotherapy, it was pointed out that the establishment of good rapport between the therapist and the patient is a sine qua non for any type of psychotherapy. Psychotherapy based on personality study requires the patient's willingness and readiness for a determined effort to take an active part in the treatment. He must be willing to discuss not only his complaints and immediate problems but also experiences and events of the more or less remote past. It is reasonable to admit a priori that the patient will not care to bring forth certain of his experiences, that he will try to omit certain events, that he will be cautious to avoid discussion of situations which might lead to revelation of material which he wants to remain in oblivion, or he will not discuss certain events for the reason that he is not fully aware of them. If the patient is brought to recognize that his being an active partner in the discussions directed by the therapist is essential for the success of the treatment, it is to be expected that he will get interested

in such discussions. One can hardly expect, however, that the patient's interest will be greatly aroused if the therapist takes a detached matter-of-fact attitude without impressing the patient that he is vitally interested—as he actually should be—in the topics he or the patient brings forward for discussion. It follows that the taking of the history marks the beginning in the establishment of the necessary adequate patient-physician relationship and traces the pathway for its further development. The scope of the history is much wider; it gives the patient the opportunity, in recounting the story of his life, to become aware of and feel certain experiences which prove to be significantly revealing to the patient himself; it also opens avenues for further therapeutic sessions, insofar as certain events, personal experiences brought forth in the history, may be taken up for more comprehensive discussions with the patient for the purpose of treatment. Thus, the taking of the history is part and parcel of the personality study and the first step in the psychotherapeutic situation.

The discussion in subsequent psychotherapeutic sessions of the mode of application of the general principles thus far dealt with is beyond the scope of this paper.

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IMPENDING MYOCARDIAL INFARCTION *

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In 1937 Feil,[1] and Sampson and Eliaser [2] described "preliminary" or "premonitory" pain in 15 (50 per cent) and 29 (48.1 per cent) † of their cases of acute myocardial infarction.

Since this subject is deemed of considerable importance, we are recording our experience and impressions. Of 61 consecutive cases of acute infarction treated in the past two years, 17 had premonitory symptoms. They are summarized in table 1. Additional data are given in 5 cases.

The symptoms have the following characteristics:

1. There may be one or more episodes of spontaneous prolonged cardiac pain, mild or severe, with or without a preceding history of coronary artery disease.
2. Cardiac pain of short duration, on customary or no exertion, suddenly appears where none existed before.
3. In patients with preëxisting angina pectoris, cardiac pain appears on rapidly decreasing amounts of exertion or at rest.

Symptoms precede infarction by a few hours, days, or weeks. Individual attacks of pain last minutes to hours. In one instance, pain lasted seven days. The attacks are considered to be the result of ischemia caused by occlusion or rapid narrowing of a coronary artery, with probable intermittent reflex vasoconstriction of other arteries to explain apparent inconsistencies in the response of pain to effort (cases 13 and 16).

During this preliminary period, physical examination, blood pressure, temperature, and leukocyte count are unchanged. Electrocardiographic abnormalities may be found, which may be due to previous myocardial damage of some duration; but there may be S–T and T-wave contours suggesting recent change (figure 1; and Feil's article, case 15). The electrocardiogram may be perfectly normal. A normal record does not rule out impending myocardial infarction.

While experiencing symptoms premonitory of another infarction, two of our cases, convalescing from an acute attack, had electrocardiograms showing progressive changes toward normal until the second infarction occurred (cases 13, 14; figures 1, 2). The third tracing in figure 1 might be interpreted as showing changes suggesting fresh myocardial activity.

In three cases summarized below, we had interpreted all the symptoms as premonitory in nature and evidence of acute infarction came as a surprise (cases 4, 13, 14).

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† The validity of this percentage is questioned, since it is not based on the total number of cases, but on an unstated fraction—27 cases.

TABLE I

Case	Blood Pressure	Past History of Coronary Disease	Interval Between Onset of Preliminary Pain and Infarction	No.	Duration of Preliminary Attacks	Circumstances	Circumstances of Infarction
1	140/90	——	3 hours	1	15 min.	Sleep	After awakening
2	140/82	Effort angina 20 months	5 hours	C.20	10 min.	Began after strenuous activity; recurrent, spontaneous for 5 hours	In bed
3	120/78	Effort angina 4 years	12 hours	Many	"Brief"	Recurrent spontaneous throughout day of usual activity	Immediately after anginal seizures
4	102/62	Effort angina 2 years; worse, 3 months	2 days	2	"Brief"	Spontaneous, promptly relieved by nitroglycerine	Apparently occurred during spontaneous anginal episodes or was "silent." EKG normal one week before
5	124/86	Effort angina 1 month	2 days	C.30	3 min.	Spontaneous, recurrent for 3½ hours	At rest
6	140/100	Infarct, 1 year previously	3 days	2	4 hrs.	Sleep	Sleep. EKG normal on entry 8 hrs. after onset; similar to EKG taken 1 yr. before. Acute changes during next 3 weeks (figure 4)
7	160/98	——	5 days	4	5–10 min.	Following usual mild activity after noon meals	Sleep
8	148/84	Infarct, 15 months previously	7 days	3	15–30 min.	Usual mild activity	Sleep
9	165/110	Infarct, 2 years previously	7 days	1	7 days	Spontaneous	At rest
10	135/92	Effort angina 20 years	10 days	4	20–90 min.	Spontaneous and sleep	Sleep

TABLE I—*Continued*

Case	Blood Pressure	Past History of Coronary Disease	Interval Between Onset of Preliminary Pain and Infarction	No.	Duration of Preliminary Attacks	Circumstances	Circumstances of Infarction
11	130/80	——	12 days	Occasional	"Minutes"	Mild effort, sleep	Mild activity
12	108/70	——	20 days	12	"Brief"	Increasingly severe on mild effort	After 3 days partial restriction; died in 7 hours
13	130/80	Infarct within 10 days before	20 days (?)	Many	5–30 min.	Mild effort, spontaneous	After 10 days strict bed rest
14	140/110	Effort angina 2 years	c.1 month	Many	"Brief"	On decreasing activity; 1 spontaneous, ½ hr.	After 5 days complete bed rest
15	140–85	——	35 days	Many	"Brief"	Increasingly frequent on less exertion, occasionally spontaneous	Upon fully clothing himself; after 1 month's intermittent partial and total bed rest
16	120/80	——	35 days	3	"Minutes"	Effort and spontaneous	After partial restriction for 13 days. While undressing. Died in 60 hours
17	124/84	——	35 days	Many	10–15 min.	Increasingly frequent on less exertion. Twice during sleep.	During usual mild activity

Of the 17 cases, infarction followed five hours after strenuous activity in one (case 2); during or after usual mild activity in six (cases 1, 3, 11, 15, 16, 17); during rest in six (cases 4, 5, 9, 12, 13, 14); and during sleep in four (cases 6, 7, 8, 10).

Of two placed at complete bed rest, infarction occurred on the fifth and tenth days (cases 13, 14). Of two at bed rest with bathroom privileges, infarction occurred at rest on the first and third days (cases 4, 12). In one, infarction occurred after 13 days of moderate restriction of activity (case 16).

Of the 17, two died—seven and 60 hours after severe infarction (cases 12, 16). Of the other 44, eight died.

After acute infarction has happened, it is easy to reconstruct the premonitory symptoms and to evaluate them correctly. When confronted with a suspected case, however, the problem is more difficult. Five possibilities exist.

(1) Infarction may not develop at all, and without treatment. This may occasionally happen when there has been an isolated, prolonged, spontaneous cardiac pain in a case of angina pectoris of some standing.

(2) Infarction may have already recently occurred.

(3) Infarction may possibly be averted by treatment.

(4) Infarction will develop regardless of what is done, but conceivably may be minimized.

(5) Infarction may not develop for several weeks after the occurrence of premonitory pains, so that one's suspicions are lulled and treatment relaxed.

That infarction may sometimes be avoided by restricting activity in suspected patients is open to speculation, since there is no way of knowing that it might otherwise have occurred. We have had, however, several patients whose threatening symptoms, suggestively premonitory, subsided and it is felt that treatment may have averted infarction. The benefit frequently following a period of restricted activity in angina pectoris is well known. This probably occurs through increased arterial collateralization of affected areas.

That the severity and mortality of infarction are minimized when occurring at enforced rest is debatable.[2] An opinion may only be reached after considerable data on this subject have accumulated.

It may be asked whether complete bed rest might not, in some of these cases, slow blood flow critically in a precariously narrowed artery and precipitate thrombosis and infarction.

In our present state of knowledge, if impending infarction is suspected from the presenting symptoms, marked to complete restriction of activity is advisable, with sedation, analgesics, and vasodilators when indicated. Several weeks' rest is recommended, since it usually takes that long, according to experimental data,[3] for effective collateral vessels to develop. The nature of subsequent treatment will depend on existent symptoms.

Even with these measures infarction and death will be inevitable in some cases, because the speed of collateral growth will be unable to keep abreast of the circulatory losses being suffered through rapid narrowing or occlusion of an artery. Where cardiac pain does not respond to nitrites, the intravenous use of aminophylline, gr. 7½, is advocated in the hope of dilating vessels supplying critical areas of the myocardium and to decrease any existing vasoconstriction. We have seen the measure dramatically relieve a patient of very severe protracted anginal pain where nitroglycerine and morphine had failed.

Papaverine, gr. ½ to 1 intravenously, has been suggested for treatment of the acute phase of infarction.[4, 5] Animals given papaverine are less prone to develop regional vasoconstriction and ventricular fibrillation following coronary ligation.[6] It may prove to be of value in the treatment of prolonged cardiac pain where reflex vasoconstriction might be present.[5]

There is experimental evidence [7] that the area of acute infarction and the immediate surrounding zone of impaired nutrition may serve as an irritable focus causing vasoconstriction in other parts of the myocardium through a vagal reflex. It is thought that many deaths from acute infarction may result from ventricular fibrillation beginning in these ischemic areas.

It has been suggested on the basis of animal experimentation [7] that atropine and aminophylline be given to prevent such a fatal outcome. Although these data are not conclusive, such therapy deserves cautious trial. The theory is that atropine will cut down the intensity of the vagal reflex. Dosage recommended is 1/75 of a grain intravenously every four hours. Such doses might produce harmful effects. If there is a marked increase of rate in a badly damaged heart, the precariously balanced cardiac circulation may not be able to forestall a relative ischemia, with further infarction or fatal ventricular fibrillation. In a rare case, intravenous atropine can have an opposite effect. It has produced complete A–V block with a pulse rate of 30.[8] A marked slowing of the heart rate may also be injurious because it may decrease the existing coronary circulation and endanger the viability of areas already poorly supplied.

Case Reports

Case 13. A 70 year old ship's engineer had been perfectly well until 10 days before entry. One morning, just before starting to work, he felt a spontaneous, very strong pressure in the mid-sternal region. This lasted only five minutes. He worked as usual that day and had no discomfort. The second morning, after some customary mild exertion, he had the same feeling of mid-sternal pressure. Although less intense, it lasted 10 minutes.

Since he had the next week free, he stayed at home or took short walks. Every morning he had a 10-minute episode of moderate mid-sternal pressure. It occurred while he walked about the house, and was relieved by rest. Between attacks he felt all right.

The day before entry, he had two attacks at rest, each lasting 10 to 15 minutes. The next day, there were two bouts of pain while walking around the house. They lasted 10 and 20 minutes. At noon he walked one and one-half miles to our office. He felt well until the moment he sat down, when he had a brief, mild pain.

His heart was normal in size, sounds, and action. Blood pressure, pulse rate, temperature, leukocyte count were normal.

Since impending infarction was suspected, he was put on absolute bed rest. Electrocardiogram that day suggested a recent anterior myocardial infarct (figure 1). Sedimentation rate was 21 mm. per hour (normal, to 10 mm.).

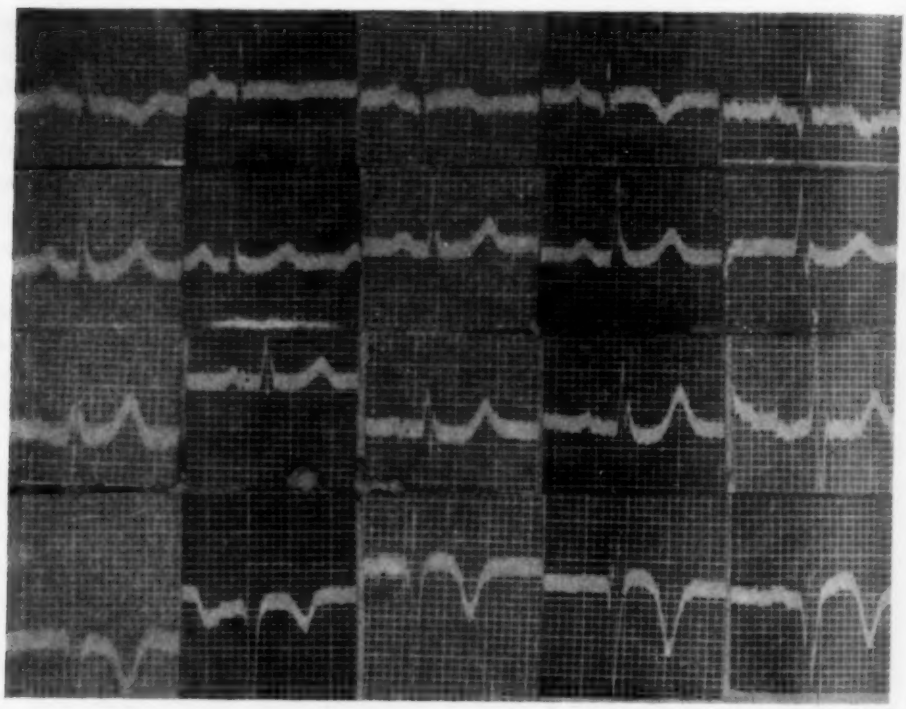

FIG. 1. Case 13: First EKG, day of entry; second EKG, sixth day of entry; third EKG, ninth day of entry, one day before second infarct; fourth EKG, thirteenth day of entry, three days after second infarct; fifth EKG, nineteenth day of entry, nine days after second infarct.

For the 10 days following hospitalization, he daily had about five mild to marked attacks of substernal pain at rest or during sleep. They lasted five to 20, rarely 30 minutes, and were very definitely and promptly relieved after taking nitroglycerine. The longer attacks occurred when the patient delayed taking nitroglycerine to see if the pain would disappear without medication. There was no fever, change in the heart sounds, blood pressure, or pulse rate. On the fifth day, electrocardiogram showed regressing T-wave changes, consistent with a healing infarct. On the eighth and ninth days he experienced several five-minute periods of interscapular aching not influenced by nitroglycerine. On the tenth day, he had two rather severe attacks of pain, one interscapular, the other substernal, for four and two hours respectively, not relieved by nitroglycerine. On the eleventh to thirteenth days he had a temperature of 37.2 to 37.6° C. Leukocyte count was 12,600. Heart, blood pressure and pulse rate remained unchanged. He was thereafter asymptomatic. Electrocardiograms showed fresh infarction.

Case 14. One year prior to admission, a 61 year old ship's cook began to have a mild steady ache in the left shoulder while working or after walking 50 yards. This would last five minutes and be relieved by rest.

After six months the pain became more intense on the same amount of exertion. It began to spread over the precordium and lower sternum.

In the last month, the pain appeared on even less work than usual, was even more intense, and lasted 10 to 15 minutes.

In the last week, he could hardly do much of anything without having pain. It was now accompanied by slight dyspnea. At no time were there any attacks at rest or in bed. There were no outstanding attacks.

He was obese. Heart sounds were distant; blood pressure was 140 mm. Hg systolic and 110 mm. diastolic. The first electrocardiogram indicated recent anterior myocardial infarction, with P–R interval .23–.24 sec. (figure 2). He was put to bed

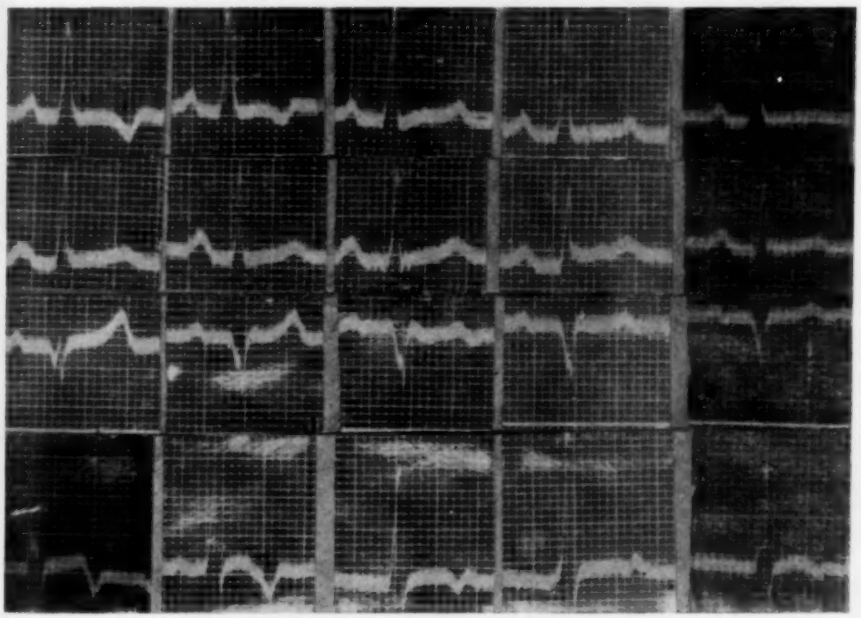

FIG. 2. Case 14: First EKG, during period of angina on exertion; second EKG, day after first spontaneous attack; third EKG, 12 days after second spontaneous attack; fourth EKG, 28 days after second attack; fifth EKG, 40 days after second attack.

with bathroom privileges, but continued to have angina pectoris if he walked about. Since we suspected impending infarction, we placed him at complete bed rest on the eighth day. On the tenth day, he had his first attack of precordial pain at rest, lasting one-half hour. On the eleventh day, electrocardiogram showed T_1 less inverted, flattening of ST_4, and a decreased P–R interval of .21 sec., consistent with healing infarct. On the twelfth day, he had bilateral gnawing shoulder pain, and distress over the precordium, lasting almost an hour. These pains were not severe and no analgesics were required. Sedimentation rate increased from 13 mm. to 21 mm. during this second week. Temperature, pulse rate and leukocyte count were normal. Blood pressure gradually dropped to 118 mm. Hg systolic and 88 mm. diastolic. A third electrocardiogram showed a posterior infarction modifying the anterior pattern. The P–R interval had increased to .24 sec.

Comment: In cases of angina pectoris where attacks rapidly become more easily precipitated, existing or imminent infarction should be considered. Infarction may occur without fever, leukocytosis, or dramatic symptoms.

Case 16. Five and a half weeks before entry, a 59 year old ship's cook had sharp aching precordial pain lasting a few minutes. It came after running for a street car. He felt well enough to continue his work, but two nights later was awakened by a similar brief attack of pain. Two weeks before we saw him he stopped working because of sluggishness, weakness, dyspnea on exertion. At home he felt quite well until the day of entry, when he had another attack at rest similar to the others.

Examination was completely negative. Electrocardiograms showed low voltage, inverted T_2 and T_3 (figure 3). Since impending infarction was suspected, he was

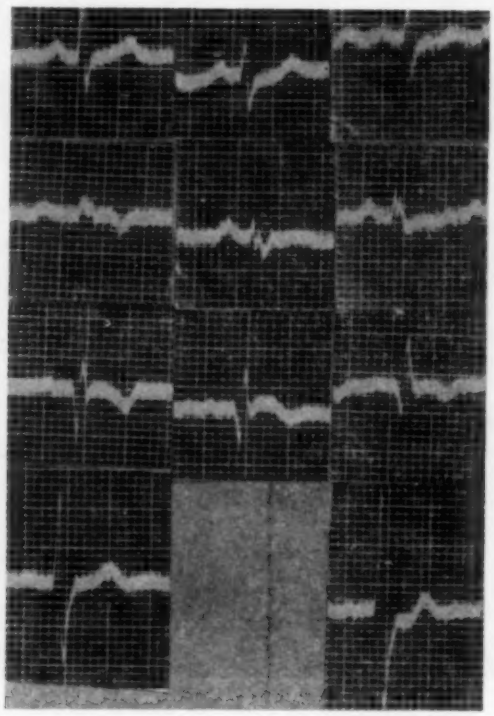

FIG. 3. Case 16: First EKG, before exercise; second EKG, after exercise until breathless, no pain; third EKG, 11 days later, following acute infarction.

observed for 10 days, ambulatory and asymptomatic. He was exercised until breathless without experiencing any pain, but there were produced electrocardiographic changes very similar to the pattern of his subsequent infarct. He was discharged and continued to rest, confined to his home. Three days later, while undressing, he suffered a very severe myocardial infarction and died in 60 hours.

Case 4. A 63 year old seaman first noted very mild pressure over the lower right chest brought on by exercise or a heavy meal. This continued for two years, and for the preceding three months he had increasingly marked pressure on decreasing exertion and after a large meal. It now began to be felt over the lower left chest and lower sternum also. Finally, he would have severe symptoms several times a day after any kind of exertion. Rest would bring relief, and further exertion would cause a recurrence of pain.

One week before entry, *an electrocardiogram was taken and found normal.* Because of the frequency and persistence of symptoms he entered the hospital.

On examination, the heart was found normal; blood pressure was 102 mm. Hg systolic and 62 mm. diastolic; leukocyte count was 8500.

On the day of entry, while sitting by his bed, he had a typical brief pain which subsided shortly after taking nitroglycerine, gr. 1/100. The next day he had a similar spontaneous pain seemingly relieved by nitroglycerine. Two days later, temperature rose to 37.4° C., and persisted for three weeks. Coupling appeared. Electrocardiograms taken after the appearance of these features showed acute changes of myocardial infarction.

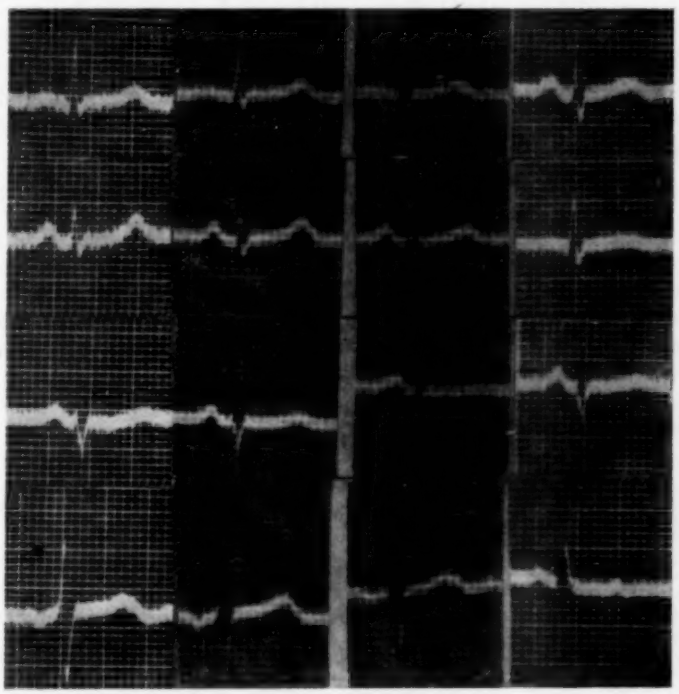

FIG. 4. Case 6: First EKG, one year before infarct; second EKG, day of infarct; after eight hours of pain; third EKG, seven days after infarct; fourth EKG, 20 days after infarct.

Case 9. An unusual case of preliminary pain in the right arm for seven days before acute infarction.

One week before entry, a 54 year old seaman developed a gnawing ache over the ventral surface of the right forearm and biceps. It was rather persistent and gradually became worse. A few hours before entry, while he was resting in bed, the pain became excruciating and he noted slight substernal pain.

When seen, he was weeping and beating his arm on the pillow to "knock out the pain." The next day, the chest pain became more pronounced. He developed fever, marked leukocytosis, pericarditis, ventricular tachycardia, and electrocardiographic changes of acute posterior infarction.

COMMENT

Acute myocardial infarction is preceded by premonitory symptoms in a goodly percentage of cases.

In a patient previously well cardiac pain, however brief and mild, sud-

denly appearing during rest or customary activity, may imply existing or imminent mycardial infarction.

In a case of preëxisting angina pectoris, cardiac pain, more readily precipitated by effort or beginning to occur at rest, may imply existing or imminent myocardial infarction.

One or more episodes of spontaneous prolonged cardiac pain may precede myocardial infarction.

In considering symptoms suspected as premonitory, it must be recognized that myocardial infarction does not inevitably follow them; but the strong possibility that it may should lead to heightened suspicion and therapeutic precautions.

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A CLINICO-PATHOLOGIC STUDY OF 100 CASES OF ACUTE AND CHRONIC GALL-BLADDER DISEASE *

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For the purpose of correlating the clinical manifestations and morphologic changes in gall-bladder disease, we studied the case histories of 100 patients who had been operated upon during the last 10 years.

There were 81 women and 19 men. The average age was 46.4 years. Three patients were over 70 and the youngest was a girl of 15. Table 1 shows the incidence according to age.

In 71 patients calculi were present, and in 29 the disease was non-calculous in type. Eighty-six patients were admitted during a more or less quiescent interval. Fourteen showed acute symptoms at the time of admission consisting of tenderness, a palpable mass in the right upper quadrant, fever and leukocytosis. At operation 11 of the 14 had calculi.

Table I

Incidence According to Age

Age	Calculous Series		Noncalculous Series	
	Males	Females	Males	Females
10–19 yrs.		2		
20–29 yrs.		3		3
30–39 yrs.	2	15	2	4
40–49 yrs.	3	16	3	4
50–59 yrs.	3	16	4	6
60–69 yrs.	2	7		2
70–80 yrs.		2		1

Etiology. Gall-bladder disease is essentially a chronic disease. Although acute cholecystitis does occur, most of the so-called acute cases are either recurrent or acute exacerbations of infection in a chronically diseased gall-bladder which has caused trouble for some time.

Infection may reach the gall-bladder through various routes. It may ascend from the duodenum by way of the papilla of Vater. Following influenza and pneumonia the infection may enter the gall-bladder through the hepatic route, whereas the portal route may carry infection after typhoid fever and colon bacilli infection. When an inflamed colon or duodenum is in direct contact with the gall-bladder the infection may enter by direct extension, and the infection may spread from the liver or the pancreas by way of the lymph stream.

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Pregnancy is an etiologic factor because of the interference with the normal emptying of the gall-bladder. It has been shown in animal experiments that only when the uterus is empty will the gall-bladder respond to the administration of fat. Although none of our patients was pregnant at the time of admission, 45 of the calculous series and 18 of the non-calculous gave a history of one or more pregnancies.

Typhoid fever as an etiologic factor is becoming less common. A history of typhoid fever was elicited in only two patients and in none of the cases was it possible to recover typhoid bacilli from the bile.

Symptoms. The difference between the symptoms of the two types of gall-bladder disease is only one of degree and not of kind. The cholecystitis is the essential condition. The calculi are only incidental and aggravate an existing inflammation, but in themselves are not capable of producing cholecystitis, as inflammation is due to infection with microörganisms. The longer the cholecystitis has been present, the more likely it is to be complicated by calculi. McEvedy found that calculi are absent in 20 per cent of patients with definite cholecystitis. McCarty, in studying 21,523 cases at the Mayo Clinic, found that only 65 per cent of the surgically removed gall-bladders contained stones. In our 86 patients with chronic gall-bladder disease, calculi were present in 60.

Analyzing the symptoms present in this group of 86 cases we find distention, flatulence, belching, nausea and vomiting, loss of weight, fever in a negligible percentage of the chronic cases, jaundice, and the vague complaints of indigestion, discomfort in the epigastrium, and sour taste. Table 2 shows the distribution of these symptoms in the calculous and non-calculous cases.

TABLE II

Relative Incidence of Signs and Symptoms in 86 Cases of Chronic Calculous and Noncalculous Cholecystitis

(60 patients had concrements; 26 had stoneless gall-bladders)

Symptoms	Calculous Series		Noncalculous Series	
	No.	Per Cent	No.	Per Cent
Distention and belching	20	33.33	19	73.07
Constipation	14	23.33	7	26.92
Vomiting and nausea	45	75.00	10	38.46
Colics	40	66.66	10	38.46
Jaundice	19	31.66	4	15.38
Typical symptoms of gall-bladder disease	45	75.00	12	46.15
Vague symptoms of abdominal distress	5	8.33	7	26.92

Cholecystography. In our experience cholecystography shows accurate diagnosis in well over 90 per cent of the cases. Graham and Cole state that cholecystography is merely a method of studying the functional activity of the gall-bladder rather than a means of indicating the exact pathologic lesions

present. It does not give the final diagnosis and is only to be considered as a contributory aid for the diagnosis of gall-bladder disease. The symptoms are more reliable than the roentgenologic report of a pathologic gall-bladder. The cholecystographic findings in relation to the pathologic lesions found at operation are frequently disappointing. In most of the cases in which visualization showed a delayed function or a faint shadow of the gall-bladder the cystic duct was patent and the gall-bladder contained bile. Occasionally the existence of diabetes mellitus, penetrating duodenal ulcer, or simple spastic conditions of the gastrointestinal tract may produce false results of the Graham-Cole test. Surgical exploration should be advised in cases in which the clinical history of gall-bladder disease is questionable even though the visualization tests suggest a normal function.

Forty-one cases of our series of chronic cholecystitis studied by cholecystography showed a poor or nonfunctioning gall-bladder, and of these 33 were distinctly pathologic at operation, three were slightly so, and two showed indefinite changes. On the other hand, three cases showed negative results of the visualization test but definite disease at operation, such as thickening and scarring of the gall-bladder wall with or without pericholecystic adhesions.

The error of the cholecystographic diagnosis was greatest in the cases with either faint shadow or delayed function without stones, whereas the Graham-Cole test proved correct in those cases in which there was a faint shadow or no visualization of the gall-bladder with distinct evidence of calculi.

Pathology. Pathologists are not in entire agreement as to what is a normal gall-bladder. It is estimated that 30 to 50 per cent of all adults over 30 years of age have chronic cholecystitis in one form or another. It is a well known fact, confirmed at autopsy, that many patients with definite disease of the gall-bladder never had symptoms. The normal gall-bladder shows lymphocytic infiltration. Lymphoid follicles in the submucosa may be numerous and are commonly found.

TABLE III

Relation of Cholecystographic Results to Pathologic Findings in Gall-Bladders Operated on as Chronic Cholecystitis

Cholecystographic Findings	Definite Lesions		Indefinite Lesions		Normal Gall-Bladders	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Poor or nonfunctioning gall-bladders	33	80.48	3	7.32	2	4.88
Normal functioning gall-bladders	3	7.32				

In our series, 47 chronically diseased gall-bladders were removed at operation and examined histologically; 25 contained stones and 11 were free from calculi. In both varieties we could distinguish three types of gall-

bladders: (*a*) those which showed signs of clinical activity; (*b*) those removed during the quiescent intervals, and (*c*) those removed because of vague symptoms of pain in the upper abdomen and indigestion over a long period of time. In these cases there was a certain asymmetry of the pathologic process. In a considerable number we observed a patchy distribution of the inflamed areas. Inflammatory changes were usually more advanced in the region of the fundus and in the body of the gall-bladder; they were more pronounced in the areas adjacent to the liver than in the free portions of the wall, which seems to confirm the theory of spreading of the infection by direct continuity from the liver.

True ulcers of the gall-bladder are very rare, and we found only three in our series, all occurring in the acute cases with calculi.

The histologic examination of the chronically diseased gall-bladders proved that the microscopic changes are very seldom an accurate index of the severity of the symptoms. Gall-bladders which were supposed to be responsible for the most aggravating symptoms of cholecystitis showed almost no change at microscopic examination. Lymphocytic infiltration is no criterion of chronic cholecystitis; small round cells may be present in the normal gall-bladder wall even in childhood. They may be found as small solitary lymph follicles or they may be diffusely distributed throughout the wall, especially in the submucosa, probably as a part of a general lymphatic hyperplasia.

The thickening of the gall-bladder wall may be extreme in one degree; in one of our specimens the wall was 1.5 cm. thick. In some cases the thickening is due to fibrosis; the connective tissue replaces the muscular and elastic tissues and the resulting scar tissue converts the gall-bladder into an inert sac containing an extremely contracted cavity. In the majority of our cases, however, the gross specimen appeared markedly thickened, whereas on section the mucosa and muscularis appeared quite normal. The thickening was due to a marked edema of the subserosa. In some of our specimens fat was collected in the subserous and muscular layers, a factor which also contributed to the apparent extreme thickening of the gall-bladder wall.

Of the 12 cases which on gross examination had marked thickening of the wall, only one showed fibrosis of all coats of the gall-bladder, the other 11 exhibiting marked edema in the subserosa. All the specimens contained calculi, usually large solitary stones. We rarely found a considerable number of smaller concrements in that type of gall-bladder disease. Sometimes a gall-bladder contracted markedly after fixation simulating a gross thickening of its wall, a feature which was clarified at histologic examination.

The epithelium of the gall-bladders, as a rule, showed no change. As Boyd points out, it is remarkable how intact the surface epithelium is even in the worst looking gall-bladders. Only in the three specimens where ulcers were found could desquamation and destruction of large areas of epithelium be observed.

Five of the examined gall-bladders had a normal mucosa and showed no marked evidence of inflammatory reaction.

With the exception of some chronically diseased gall-bladders which showed an edematous and markedly swollen wall with dense lymphocytic infiltration, abundant formation of granulation tissue and thickened swollen villi, in the subacute or quiescent cases, the remaining 35 specimens were divided into two groups: (*a*) those with slight lymphocytic infiltration where a small number of lymphocytes was scattered throughout the mucosa and partly through the inner layers of the muscular coat, and (*b*) those cases with marked diffuse or focal small round cell infiltration throughout the wall. The infiltrating cells were lymphocytes, plasma cells and cells of wandering mononuclear type.

Fourteen cases were only slightly infiltrated with lymphocytes, whereas 21 exhibited marked small round cell infiltration. The calculous as well as the noncalculous variety were almost equally distributed in either group, a fact which again proved that stones are not essential in the production of cholecystitis but that their presence may only be an aggravating factor in the existing inflammation.

Rokitansky-Aschoff sinuses were observed in a high percentage of our cases. The hernia-like outpouchings of the mucosa frequently passed through all coats of the wall, here and there forming small dilated bags in the subserosa or in the serosa. Since they were found in the normal as well as the diseased gall-bladders, they certainly do not indicate the severity of the cholecystitis. We observed, however, that the Rokitansky-Aschoff sinuses were rather shallow in all cases where the muscular coat was hypertrophic.

In nearly all the specimens the submucosa showed an increase of the connective tissue and the walls were only slightly infiltrated by small round cells, even where the subepithelial mucosa was densely infiltrated by lymphocytes.

The muscularis and subserosa showed considerable change and were also involved in the cases in which the mucosa apparently was quite normal. The muscular coat rarely takes an important part in the thickening of the gall-bladder wall. We found hypertrophy of the muscularis in four cases, two of the calculous type and two of the noncalculous variety, which suggests that hypertrophy of the muscularis is not necessarily associated with the presence of stones. In our cases, the hypertrophy of the muscular coat was manifested by an increase in the size and to a certain degree in the amount of the smooth muscle cells. As a rule, the thickening of the muscularis was more prominent in the mid-portion of the gall-bladder than on both ends.

Edema, though never as outspoken as in the serosa, was found in seven of our chronic cases, showing a slight spreading of the smooth muscle fibers and commonly exhibiting a diffuse infiltration of small round cells. The edema of the muscular coat when present always was associated with an extensive edema of the subserosa which is one of the outstanding features of the chronically inflamed, thickened gall-bladders, as observed in all specimens which at gross examination revealed a marked thickening of the wall. Except for edema, the characteristic lesions in the subserosa were dilated capil-

laries and lymphatics, areas of hemorrhage, and focal collection or diffuse infiltration of chronic inflammatory cells. In our five cases with much involvement of the serosa, two were of the noncalculous and three of the calculous variety.

Five cases of hydrops were found, all with stones. The histologic examination showed a rather thin wall. The mucosa was intact but atrophied, showing only a small number of short sessile villi. The muscular as well as the sub-serous coat revealed a high degree of atrophy, and the wall was diffusely infiltrated by a relatively small number of chronic inflammatory cells and fibroblasts. The contents were sterile.

If a pyogenic infection is superimposed, empyema of the gall-bladder may develop, a complication we found in four cases of the calculous type. Histologically, there was thickening, especially of the muscular and subserous coats. The mucosa contained gangrenous patches with sloughing off of varying sized areas. The wall was diffusely infiltrated by numerous inflammatory cells and showed hyperemia as well as dilatation of the lymphatics and pronounced edema of the subserosa. In one case the vascular supply was damaged, producing thrombosis of the arteries of the submucosa followed by necrosis and gangrene of the mucosa.

Cultures were sterile in two of the four empyemata. In a third case we found staphylococci and *B. coli* in comparatively small numbers.

Seven of our histologically examined gall-bladders were acutely inflamed and of the calculous variety. The inflammation probably was due to an acute obstruction of the cystic duct. The characteristic features were edema especially in the outer coat and diffuse infiltration of the wall with lymphocytes and polymorphonuclear leukocytes which were most marked in the muscularis and subserosa. Compared to acute inflammation of the appendix, it is remarkable that the gall-bladder is infiltrated by a relatively small number of polymorphonuclear leukocytes and that the lymphoid elements showing diffuse or focal infiltration in some of our cases were the predominant inflammatory cells. The gall-bladder wall showed distended blood vessels and lymphatics as well as patches of recent hemorrhage. In one specimen there were multiple small abscesses in the wall. The surface epithelium showed large areas of desquamation.

The pathologically acutely inflamed gall-bladder is not always identical with the clinically acute cholecystitis. Not infrequently after the subsidence of fever, leukocytosis, pain and tenderness, the gall-bladder removed at operation may show evidence of acute inflammation. On the other hand, the histologic feature of recovery of acute cholecystitis is the appearance of eosinophiles in the gall-bladder wall, as we found in two cases. The clinical symptoms in these cases had subsided 10 days before operation. Besides the finding of eosinophiles the microscopic examination revealed the signs of chronic inflammation and repair, thus confirming the clinical symptoms.

The appearance of lipoid in the gall-bladder has created widespread discussion. Mentzer showed that cholesterosis is present in 37 per cent of

gall-bladders seen at necropsy and in 22 per cent of surgical specimens. In our series we found 10 lipoid gall-bladders, seven of the calculous and three of the noncalculous variety. The lipoid deposit, which is an ester of cholesterol, in most cases is confined to the epithelial cells in the tips of the villi of the mucosa of the gall-bladder, lying for the most part at the base of the cells. In two of our gall-bladders it was scattered throughout the stroma. The lipoid in the gall-bladder, as a rule, is of no practical importance, although it may be the first stage of an aseptic process which can eventually set free concretions of cholesterin, thus forming a nucleus for further deposits. Although it is often an additional finding in chronic cholecystitis, it is likewise found in normal specimens. Aschoff and Boyd found it as frequently in the normal as in the diseased gall-bladder.

Gall-bladder disease is quite often associated with involvement of the liver. In our series the liver was affected in eight of the calculous cases and one of the noncalculous, the involvement varying from a thickening of the capsule to enlargement, mottling of the liver and abscess formation.

The pancreas may be affected by direct extension as well as by the hematogenous or lymphatic route. In our series the pancreas showed enlargement and diffuse fibrosis in five cases. In none of the cases showing involvement of the liver or pancreas was a microscopic examination of the diseased organs done.

The appendix was removed in 37 cases at the time of cholecystectomy. The inflammatory changes in 32 appendices examined microscopically are to be considered as coincidental, as many of them were either definitely normal or showed approximately the same percentage of chronically diseased appendices as found on microscopic examination of appendices studied at autopsy.

Pericholecystic adhesions were present in 39 cases, 24 calculous and 15 noncalculous. They varied from slight adhesions of the fundus to the duodenum to extensive fibrous adhesions involving the whole gall-bladder as well. They apparently had no bearing on the preoperative symptoms as quite a number of patients who complained of severe and frequent biliary attacks showed none or just a few adhesions at operation. The postoperative course also revealed no connection with the presence or extent of the pericholecystic adhesions.

Table 4 shows additional operative findings in the 86 cases.

Final Results. Follow-up reports were received from 72 patients, 65 with chronic cholecystitis and seven with acute inflammation. The period of observation following discharge from the hospital varied from five months to nine years.

Of the 65 patients with chronic cholecystitis, 35 of the 45 who had stones were either permanently cured or greatly improved, four had only temporary complete relief, and six showed no improvement at all. Of the 20 patients in the noncalculous group, 13 reported permanent relief, three had only temporary complete relief, and four were made worse by the operation.

TABLE IV

Additional Operative Findings of Chronic Calculous and Noncalculous Cholecystitis
(60 patients had concrements, 26 stoneless gall-bladders)

Findings	Calculous Series		Noncalculous Series	
	No.	Per Cent	No.	Per Cent
Pericholecystic adhesions	24	40.00	15	57.69
Hepatitis	8	13.33	1	3.84
Pancreatitis	5	8.33	—	—
Enlarged portal glands	5	8.33	3	11.52
Appendix				
Normal	5	8.33	2	7 68
Acute inflammation	1	1.66	—	—
Chronic inflammation	17	28.33	—	—

In both groups the ones showing only temporary relief reported recurrence of symptoms from three months to one year after cholecystectomy.

The seven patients with acute cholecystitis from whom final reports were received were either completely cured or greatly improved.

Regardless of how long the patient had had symptoms, roughly 75 per cent obtained good results from cholecystectomy and approximately 25 per cent were no better after operation than before.

From histologic examination of the gall-bladder in the cases with good postoperative results, it appears that the removal of the gall-bladders which presented definite pathologic changes was followed by better clinical results than cholecystectomy in cases which showed only minimal pathologic changes in the gall-bladder.

Table 5 shows the final results in this series of 100 cases.

TABLE V

Postoperative Results According to Follow-Up Reports

Results	Calculous Series				Noncalculous Series			
	Acute		Chronic		Acute		Chronic	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Cured	4	36.36	30	50.00	1	33.33	8	30.76
Improved	2	18.18	5	8.33	—	—	5	19.23
Temporarily improved	—	—	4	6.66	—	—	3	11.53
Unimproved or worse	—	—	6	10.00	—	—	4	15.38
No reply	5	—	15	—	2	—	6	—
Total	11		60		3		26	

CONCLUSIONS

From this study it would appear that noncalculous cholecystitis is best handled by medical means. When this management does not adequately relieve the patient cholecystectomy is recommended.

In the calculous type surgery is the treatment of choice.

Improvement in the results of treatment of gall-bladder disease requires a better knowledge and understanding of the physiology of the gall-bladder and biliary tract than we possess at this time.

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SUBCLINICAL VITAMIN DEFICIENCY
V. THE ASSAY OF SUBCLINICAL THIAMIN DEFICIENCY *

By Mildred Carleen Hulse, M.A., Norman Weissman, Ph.D., Elmer Stotz, Ph.D., Marshall Clinton, M.D., Joseph W. Ferrebee, M.D., *Boston, Massachusetts*

Introduction

The assay of subclinical thiamin deficiency is primarily a problem in defining changes in thiamin nutrition before signs or symptoms of deficiency make their appearance. Accomplishment of this end presupposes a knowledge of normal thiamin nutrition; that is, normal extracellular and intracellular thiamin concentrations, and the size and sequence of variation in these values which may precede the occurrence of clinical deficiency.[1] The purpose of the present study is to determine whether measurable changes in either extracellular or intracellular thiamin concentrations occur in subclinical thiamin deficiency. For comparison, observations are also reported on thiamin excretion and on pyruvate metabolism.

Method

Six normal male medical students, 20 to 25 years of age, were placed on a thiamin deficient diet of 2700 to 3300 calories (2000 non-fat) containing, by calculation and by analysis, less than 0.2 milligram of thiamin. Supplements of vitamins A, D, C, riboflavin, and niacin were supplied daily and, in addition, four of the subjects were given gelatin capsules and told that the capsules would contain either lactose or thiamin.

A gauge to changes in cellular thiamin concentrations was obtained by measuring the yeast stimulating activity of samples of skeletal muscle removed from the gluteal region with the Silverman biopsy needle.[1, 2] Samples were removed fasting in the early morning. The yeast fermentation method of thiamin assay [2] was used without correction for sulfite blanks.[3] The estimates of thiamin concentration, therefore, exceed the true values [4] and are useful chiefly in showing the decrease that occurs during subclinical deficiency. Estimation of changes in extracellular thiamin concentration was obtained in a similar manner by following the yeast stimulating activity of samples of fasting blood plasma.[5]

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This report is a preliminary study whose completion has been delayed by other work related to the war effort.

Since muscle thiamin is predominantly intracellular, its concentration was expressed in terms of another predominantly intracellular constituent, namely phosphorus, rather than in terms of wet weight of muscle.[4] The thiamin concentrations so expressed are relatively independent of possible variations in the proportion of extracellular and intracellular phase obtained in the small muscle biopsies. Muscle phosphorus was determined in each biopsy sample by the Kuttner-Lichtenstein technic[6] of phosphorus estimation.*

The urinary excretion of thiamin was determined by the thiochrome method.[7] Tolerance tests for the estimation of thiamin deficiency by measurement of thiamin excretion were performed according to the procedure of Robinson, et al.[8]

Blood pyruvate concentrations[9] were measured fasting, before, immediately after, and on recovery from a standardized exercise,[10] and before and after the ingestion of 50 grams of glucose.[11] Blood lactate concentrations were determined on the same samples.[12] Air samples were analyzed in duplicate for carbon dioxide and oxygen content on a Haldane-Henderson gas analyser. The exercise used was a 10 minute walk on a 3 mile an hour treadmill with an 8 degree slope.

General clinical condition was estimated from routine inquiry and observation and from the daily measurement of weight. Four of the subjects were allowed to continue their usual school work. Two were asked to increase their physical activity and for this purpose walked 15 to 20 miles a day.

The experiments were performed in late fall and early winter and the following observations were made.

Results

The subjects complained of the monotony of their diet almost from the first day; they did not, however, lose appetite and left to their own wishes managed in all but one instance to eat enough to maintain weight. The subject, Ki, who walked 15 to 20 miles each day over a period of 18 days, lost 3 kilos, chiefly in the last portion of this period.

During the first week of thiamin deprivation neither the subjects nor the examiners were able to convince themselves that symptoms of deficiency were making their appearance. Minor fluctuations in sense of well-being were distinguished but were of short duration and erratic occurrence. In contrast, during the latter part of the second week each of the four subjects continuing to that time felt and looked dispirited, whether from monotony of diet or other cause they could not say. On the fifteenth and sixteenth days of the deficient régime two of the subjects, Ki and Ko, received 2 milligrams of thiamin in their daily lactose "placebo" capsule. Since they were unaware of this addition, it is significant that each experienced within about 18 hours a definite improvement in sense of well-being.

* We are indebted to Miss Virginia Rowland for the phosphorus analyses and to Miss Marion Brian for her preparation and supervision of the diets.

As has been previously observed,[13, 14, 15] the urinary excretion of thiamin decreased promptly when the subjects were placed upon the deficient diet. Instead of normal values of the order of 100 micrograms per 24 hours, values of the order of 25 to 50 micrograms were observed at the end of a few days of thiamin deprivation. The excretion of thiamin at the end of two weeks' deficiency was less in subject Ki who exercised than in subject Ko who did not; 23 micrograms per 24 hours for Ki, compared to 47 micrograms for Ko.[16] Tolerance tests at the end of two weeks' deficiency showed that in subjects F and L, 6 and 8 per cent respectively of the 5 milligrams of thiamin administered with the noon meal was excreted in 24 hours. The lower limit of normal chosen by Robinson et al. for this test was an excretion of 7 per cent.[8]

TABLE I

Effect of Thiamin Deficient Diet upon Yeast Stimulating Activity of Plasma of Normal Men

(Values expressed in thiamin equivalents, millimicrograms per milliliter of plasma)

Subject	Control Value	Deficient Diet		Normal Diet	Remarks
		7 days	18 days	4 days	
Ko	6.0	—	3.9	4.8	Usual exercise
Ki	4.9	—	2.9	4.5	Walked 15–20 miles a day
S	6.1	4.1	—	—	Usual exercise
B	5.4	2.5	—	—	Walked 15–20 miles a day

The observations on plasma are given in table 1. The yeast stimulating activity of plasma decreased on the deficient diet and returned toward normal with the resumption of a normal diet. The decrease in plasma activity was more marked in the patients who exercised.

TABLE II

Effect of Thiamin Deficient Diet upon Yeast Stimulating Activity of Skeletal Muscle

(Values expressed in thiamin equivalents, micrograms per milligram of muscle phosphorus)

Subject	Normal Control	After 18 Days of Deficient Diet	Remarks
L	.41	.29	Usual exercise
F	.35	.28	Restricted exercise
Ko	.43	.28	Usual exercise
Ki	.37	.27	Walked 15–20 miles a day
S	.48	—	Usual exercise
B	.32	—	Walked 15–20 miles a day

The observations on muscle are given in table 2. The higher control values were observed in the individuals who had the higher plasma values, Ko and S. There is evidence that the differences in control values reflected differences in recent intake of thiamin.[17] Subject S, who had the highest value, 0.48, had eaten a quantity of chicken and nuts at midnight, eight

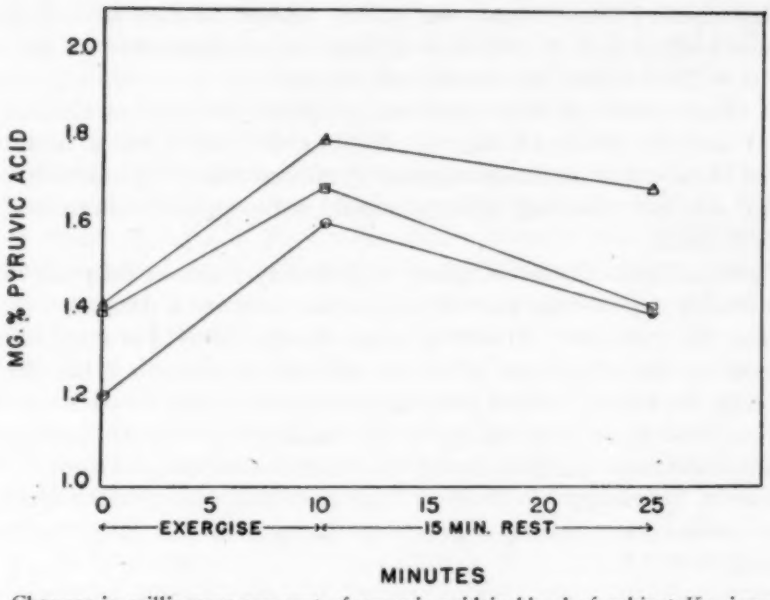

FIG. 1. Changes in milligrams per cent of pyruvic acid in blood of subject Ko, immediately after, and 15 minutes after 10 minute exercise.
○ = Normal control
□ = After 7 days on deficient diet
△ = After 18 days on deficient diet

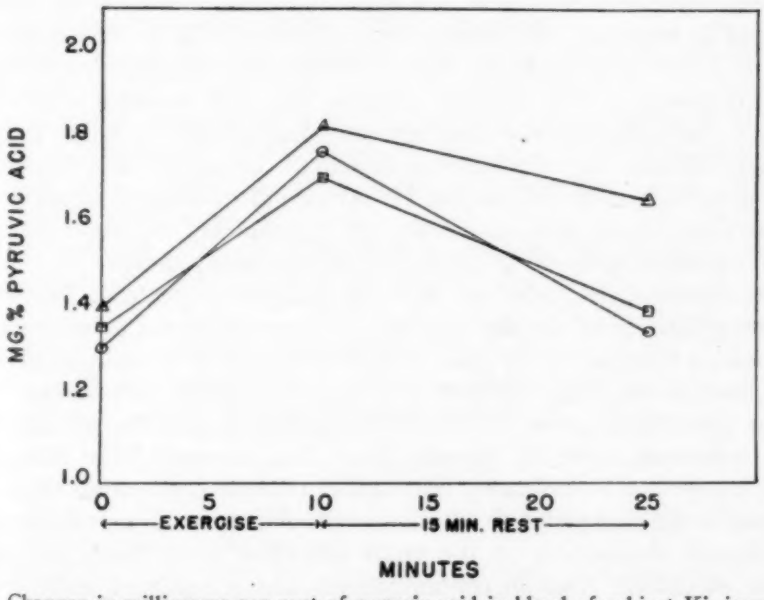

FIG. 2. Changes in milligrams per cent of pyruvic acid in blood of subject Ki, immediately after, and 15 minutes after 10 minute exercise.
○ = Normal control
□ = After 7 days on deficient diet
△ = After 18 days on deficient diet

hours before the control biopsy was taken. After 18 days of deficient diet a common level of 0.28 ± 0.01 was reached, quite independent of the control values or of the amount of exercise performed.

The observations on blood pyruvate are given for patients Ko and Ki in figures 1 and 2. After 18 days of deficient diet there was a delay in the return of blood pyruvate concentrations to normal following exercise. Similar delay was not observed after stimulation by glucose administration in subjects F and L.

In both subjects Ko and Ki there is definitely a slower decrease in blood pyruvate after the exercise period, which may indicate a decreased ability to metabolize the pyruvate. However, since absolute blood pyruvate values are dependent on several factors which are difficult to control, it has been suggested that the ratio of blood pyruvate to lactate would be of more critical value in assessing an impaired pyruvate metabolism.[12] Such ratios did not show any conclusive changes during the thiamin deficiency period.

Thiamin deficiency of 18 days' duration did not produce changes in oxygen consumption during exercise or changes in the daily excretion of citric acid.[18, 19, 20] *

Discussion

In the present experiments, clinical evidence of thiamin deficiency was detectable by the end of the second week of thiamin deprivation. The symptoms observed were vague, being chiefly a change in sense of well-being that could be improved by thiamin administration. As in the experience of Williams, et al.[13] the symptoms were sufficient for recognition in a carefully controlled group of experimental subjects, but it is doubtful whether they could have been distinguished and correctly interpreted in a single individual presenting himself for medical examination. It seems fair, therefore, to apply the term "subclinical" to the 18 day period of thiamin deficiency used in these experiments, and to discuss as "subclinical" the abnormalities of thiamin nutrition observed by means of the laboratory assays.

The abnormalities observed were as follows: a decrease in the yeast stimulating activity of samples of plasma, a decrease in the urinary excretion of thiamin, a decrease in the yeast stimulating activity of samples of skeletal muscle, and an impaired ability to metabolize pyruvate. Assuming for the moment that the decrease in the yeast stimulating activity of plasma and muscle represents losses of thiamin from these tissues,[4, 5] the observations may be interpreted as indicating that during thiamin deprivation there occurs a decrease in the concentration of thiamin circulating in the extracellular fluid, a consequent diminution of the renal excretion of thiamin, an eventual decrease in cellular thiamin concentrations, and a resultant impairment of pyruvate metabolism. All these changes occur to a measurable degree in the

* We are indebted to Dr. Martin L. Deakins of the Laboratory of Dental Medicine for the citric acid analyses.

period of deprivation termed "subclinical." The selection of any one of them for measurement as a criterion diagnostic of subclinical deficiency is, therefore, a matter of choice to be decided on such basis as: convenience, or availability of laboratory technic; discriminative capacity, in the sense of sensitivity to small changes in nutritional status; and specificity, or freedom from involvement in irrelevant physiologic or pathologic variables.

In general, there is reason for feeling that measurements of thiamin excretion reflect changes in the concentration of freely diffusible extracellular thiamin [5] and that measurements of blood pyruvate reflect changes in the concentration of fixed enzymatically active, intracellular thiamin.[21] The assay technics are, therefore, to a certain extent interchangeable. But since renal function also affects thiamin excretion,[22] and since variations in carbohydrate metabolism and pyruvate formation also affect blood pyruvate concentration, it is evident that the indirect assays of thiamin nutrition, namely thiamin excretion and blood pyruvate concentration, are applicable primarily to essentially normal individuals in whom normal baselines of renal function and pyruvate formation may be assumed. The direct assays of extracellular or intracellular thiamin concentration, that is, plasma and muscle analyses, on the other hand, should have a wider applicability once further experience has established their normal ranges and interrelationships.

Summary

Subclinical thiamin deficiency was induced in six normal young men by thiamin deprivation of seven to 18 days' duration.

Clinical symptoms were minimal and not in themselves sufficient for diagnosis.

Measurable changes, however, were observed in the yeast stimulating activity of samples of fasting plasma, in the urinary excretion of thiamin, in the yeast stimulating activity of samples of skeletal muscle, and in the metabolism of pyruvate.

The probable significance of these measurements and their use in the diagnosis of subclinical thiamin deficiency are discussed.

Conclusion

A measurable decrease in both extracellular and intracellular thiamin concentrations may be detected in subclinical thiamin deficiency.

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HEMOPTYSIS IN TUBERCULOSIS, WITH A DIFFERENTIAL DISCUSSION OF OTHER CAUSES *

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IF romancing were permissible in medicine, we might indulge in an interesting consideration of the sharp turns hemoptysis has wrought in the lives of important personages and in the history of the world. It often precipitates a psychological panic through the fear of impending dissolution and, then, spurns death in order to condition destiny. Allen K. Krause once said, "Sometimes a man will write a new kind of history. Its keynote will be the shaping of human destiny by disease." In such a history, hemoptysis should have a prominent place.

Hippocrates' statement that "the spitting of pus follows the spitting of blood, consumption follows the spitting of this and death follows consumption" is in line with the prevailing early belief that hemoptysis was a causative factor in tuberculosis, rather than an effect; a belief apparently not seriously questioned until the time of Richard Morton in the seventeenth century. Though Morton was doubtful as to whether hemoptysis caused tuberculosis, he advised that every patient spitting blood should be diagnosed as having consumption and that treatment should be instituted at once. In the beginning of the nineteenth century, Bayle definitely concludes that "phthisis provoked the hemoptysis, but not that it is the result of it." Andral, Louis and Villemin concurred in this conclusion. Even after Bayle, Laennec and Louis, through their clinical and pathological investigations, had greatly clarified the picture of tuberculosis and freed hemoptysis from this unjust charge, there was a temporary reversion toward the old belief that hemoptysis figured as a cause of tuberculosis, rather than an effect.

In our own day, we have witnessed a tendency on the part of many doctors to accept the spitting of blood as being virtually pathognomonic of tuberculosis, with a consequent neglect of differential diagnosis. In the light of present knowledge, such presumption is inexcusable. This is the more surprising since Aretaeus and other Greek physicians recognized many causes of blood spitting 1,800 years ago.

Considering the psychological effects of hemoptysis, Aretaeus wrote, "frightful despair sometimes seizes such persons; for really, who is there who can have a bringing up of blood without having a terrible fear of death." Although this is true, the well-poised patient may meet the issue of blood with calm fortitude and accept the shaping of destiny with resignation.

The correlation of bedside and autopsy findings, the invention of the stethoscope and the microscope, the discovery of the tubercle bacillus, the development of the roentgenogram, bronchography, bronchoscopy and col-

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lapse therapy have brought a better understanding of hemoptysis, its causes and its control.

Literally, hemoptysis means spitting of blood, but clinically, it means the expectoration of blood which comes from the larynx, the trachea, the bronchi, or the lungs. After struggling with the Greek description of such bleeding, Francis Adams considered "a bringing up" as the most literal English translation. When called to see a patient with blood spitting, the first duty of the physician is to distinguish between true hemoptysis and blood which comes from the nose, mouth, or pharynx. Time will not permit the enumeration of all the possible pathological conditions above the larynx, which may give rise to so-called spurious hemoptysis. The differential diagnosis is not always easy, and occasionally all efforts fail, at least temporarily, even with the aid of a skilled nose and throat specialist. This is particularly true when the spitting of blood is intermittent and the examining physician has only the history to guide him. Such cases should be required to report for examination during the period of blood spitting. Not infrequently blood in the throat is due to post nasal oozing, bleeding gums, or varicose veins at the base of the tongue. Patients who awaken after a night's sleep with blood in the throat, which is expectorated without cough, may have slight bleeding from the gums, or nasopharynx. Patients who give a history of slight blood spitting during the day, with no obvious cause, should be examined for varicosities, at the base of the tongue. Often, in such cases, bleeding can be induced through friction caused by voluntary muscular contraction and movement of the tongue. If, after a thorough investigation, doubt exists, a careful search for tubercle bacilli is indicated and the diagnosis should await definite pathognomonic data. These simple sources of blood spitting, located above the larynx, are stressed because they frequently cause unwarranted fear of pulmonary disease and because they may exist in patients who are at the same time suffering from manifest pulmonary tuberculosis. In either case, they may give rise to great alarm and a prompt diagnosis with positive reassurance may quickly lift the anxious victim out of a depleting psychological slump.

Before discussing true hemoptysis, malingering must be mentioned. Self-induced traumatic hemoptysis is rare in civilian practice, but among the less scrupulous inmates of impregnable prisons, it is not uncommon. Such culprits have designs upon hospitalization where escape may be less difficult. Now that we are at war it is important to remember that unstable or unscrupulous soldiers may seek the shield of chronic invalidism and government compensation, as preferable to the responsibilities of war with its inevitable hazards. Among the common methods employed are forceful gum sucking, cutting the gums or soft palate with a razor blade and traumatizing the posterior nares or the pharynx with a needle or some other sharp instrument. Constant awareness and vigilance on the part of physicians and nurses should discover the cause.

True hemoptysis is a common symptom of pulmonary tuberculosis, oc-

curring in approximately 50 per cent of all cases at some time during the course of the disease, and it may be said that in spite of increasing knowledge of other sources, tuberculosis continues to be the most common cause of this shocking symptom. It may be one of the earliest manifestations of tuberculosis, and in that event it often proves to be a genuine boon to the patient because it puts the fear of God in his heart and drives him to his physician in a receptive mood. It is more common and likely to be more profuse as the disease advances, especially in the ulcerative types. The hemorrhage may be slight, representing capillary oozing; moderately severe from eroded blood vessels; or profuse from the rupture of aneurysmal vessels into tuberculous cavities. It is the latter type which occasionally leads to sudden death. There may be a great gush of blood from the mouth, a few last gasps for breath and the exsanguinated patient lies limp and wan in his own blood when the doctor arrives. This picture may be simulated by the rupture of an aneurysm of the aorta or one of its branches, into a bronchus. If the patient lives until the doctor arrives, the prognosis is sufficiently good to justify the reassurance which is so essential in the treatment of hemoptysis. Patients may recover from profuse pulmonary hemorrhage and never bleed again, or the hemorrhage may recur from time to time with ultimate recovery or a fatal termination. Slight and moderate bleeding is more common, less dangerous and psychologically, not so shocking.

In the great majority of cases, the application of modern diagnostic methods leads to a definite diagnosis. Yet, with the mounting number of doubtful cases, augmented by what apparently represents increasing frequency of non-tuberculous broncho-pulmonary conditions, prompt differential diagnosis becomes imperative. Although it is important to make an early diagnosis when pulmonary tuberculosis is the cause, it is even more important in the acute pneumonias, bronchogenic carcinoma, lung abscess and bronchiectasis, all of which are common sources of hemoptysis. The truth of this statement becomes clear when we consider the brilliant results of early modern medical and surgical therapy in these conditions and the utter failure which may follow delay. When hemoptysis occurs in the course of manifest pulmonary tuberculosis under management, usually its source is easily determined; but when it occurs in a person not previously examined, a thorough diagnostic study is required. If pulmonary tuberculosis is found to be present, this is to be accepted as the most probable cause of the blood spitting. It must be remembered that hemoptysis may arise from tuberculous ulceration of the bronchi or trachea. As a rule, such lesions are associated with manifest pulmonary tuberculosis and, usually, acid fast bacilli are found in the sputum, even though active pulmonary tuberculosis is not demonstrable.

As a rule, the differentiation between tuberculosis and other causes of true hemoptysis is not difficult, but it must be remembered that, occasionally, tuberculosis may cause blood spitting when the disease is not definitely demonstrable, either by physical examination, or roentgenogram. Tubercle

bacilli have been found in the sputum of patients in whom physical examination and roentgenographic studies were reported as negative. In such cases, the history and the symptoms of toxemia, linked with a diligent search for acid fast bacilli, may make the diagnosis reasonably certain.

Recently Jackson and Diamond,[1] after careful diagnostic studies, reported 436 non-tuberculous cases with hemoptysis as follows: Bronchiectasis, 138; primary carcinoma of bronchus, 82; tracheobronchitis, 74; pulmonary abscess, 51; no evidence of disease, 34; non-suppurative pneumonitis, 15; suppurative pneumonitis, 11; adenoma of bronchus, 11; secondary cancer of lung, 6; lobar atelectasis, 4; primary carcinoma of trachea, 2; suppurating pneumoconiotic lymph node discharging into bronchus, 1; non-specific granuloma of bronchus, 1; streptothricosis, 1; chondroma of bronchus, 1; osteoma of trachea, 1; dermoid cyst communicating with bronchus, 1; broncholithiasis, 1; neurofibroma involving wall of bronchus, 1. Among the causes not mentioned by Jackson and Diamond [1] are: mitral stenosis; blood dyscrasias; trauma, with or without rib fracture; exploratory needling; pneumonia; infarction of the lung, embolic or thrombotic; bubonic plague; bronchial fluke; blast injuries; sporotricosis; hydatid cyst; fat embolism; cystic disease of the lung; aortic aneurysm; foreign body; arteriosclerosis of the pulmonary vessels; trichinosis and possibly strenuous physical effort and severe coughing, as in whooping cough; also, vicarious hemorrhage from interrupted menstruation has been listed as a cause.

These figures give a very good idea of the non-tuberculous sources of hemoptysis and the Jackson and Diamond series indicates the relative frequency of the more important causes. Apparently, bronchiectasis, bronchogenic carcinoma and lung abscess are more common than they used to be, but no doubt improved diagnostic methods and increased alertness on the part of the physician, account for the seeming increase in frequency. At any rate, the diagnostic problem becomes more acute and differential diagnosis more obligatory.

Unfortunately, the bronchopulmonary conditions which figure as the common causes of hemoptysis may so closely simulate tuberculosis and each other that diagnosis becomes most difficult, yet the skilled clinician knows that each of these conditions has its own distinctive clinical pattern with which he is quite familiar and from which it seldom wholly strays. If pulmonary tuberculosis is closely simulated by bronchiectasis, bronchogenic carcinoma, pulmonary abscess or tracheobronchitis, a careful comprehensive study of the history usually reveals a clue which at least temporarily classifies the condition for further diagnostic investigation. Such a tentative diagnosis serves as a guide for physical exploration and determines the judicious employment of such diagnostic procedures as successive roentgenographic films guided by fluoroscopy and supplemented by bronchography when indicated. Laboratory studies of sputum, blood examinations and finally bronchoscopic explorations, biopsies and the examination of exudates may help to clarify the diagnosis.

Bronchiectasis is the most common non-tuberculous source of hemoptysis. Without going into great detail, the following features are to be emphasized as important in the differential diagnosis: a progressive, productive cough following an acute bronchopulmonary episode, usually dating back to childhood; purulent sputum, occasionally foul smelling and persistently negative to all tests for acid fast bacilli; cough and expectoration precipitated or aggravated by lying down, stooping over or laughing; and finally, clubbing of the fingers.

Of course, all stages of bronchiectasis must be considered and those cases which are clinically less obvious and the cases of dry bronchiectasis may add to the diagnostic difficulties. The physical examination, with roentgenographic examination, bronchography and bronchoscopy as indicated to supplement the history, should lead to a definite diagnosis.

Hemoptysis from bronchogenic carcinoma should be considered when persistent, productive cough with blood streaking or frank hemorrhage develops without obvious acute bronchopulmonary episodes. The probability of carcinoma is enhanced if the cough becomes progressively worse and resists all therapeutic measures. Additional evidence may or may not be elicited by physical examination. The reduction or suppression of breath sounds on the affected side is a significant sign. Roentgenographic studies may be equally uncertain, especially in early bronchogenic carcinoma. In every case of hemoptysis unexplained after a careful diagnostic study a bronchoscopic examination should be advised and pieces of tissue secured for histological studies when possible. This is particularly true when the above symptoms dominate the history. When gross pathological changes appear it may be too late. Unfortunately, pneumonectomy is seldom feasible after hemoptysis occurs. In other words, spitting of blood is not an early sign of bronchogenic carcinoma.

Often, hemoptysis from pulmonary abscess may be diagnosed by a carefully taken history and roentgenographic studies. If there is a history of a provocative episode such as an operation about the nose, throat or mouth, bronchopneumonia or the aspiration of a foreign body followed by fever and cough, the diagnosis becomes reasonably certain. The cough may be unproductive for a few days, then gradually or suddenly give rise to purulent sputum, often foul smelling, with an unpleasant sweetish taste. If the roentgenogram shows evidence of pneumonitis with cavity formation and the sputum is persistently negative for acid fast bacilli, the diagnosis of pulmonary abscess is assured. In some cases it may be necessary to secure different roentgenographic exposures or even the employment of the Bucky technic in order to demonstrate the abscess cavity. If doubt remains bronchoscopy should be employed.

Because of the war, hemoptysis caused by rupture of the fragile lung tissue from blast either in the air or under water must be considered. The history of exposure to such an injury with associated effects upon other organs should aid in the diagnosis.

Considering our far-flung battle fronts, bronchial fluke as a cause of hemoptysis also must have consideration. It is said that casualties from the Pacific presenting puzzling blood spitting have been observed in San Francisco Hospitals and that, after some delay, it was found that the hemoptysis was due to bronchial fluke apparently acquired from pickled crab meat captured from the Japanese. After some delay, the eggs of the fluke were found in the sputum, thus confirming the diagnosis.

Hemoptysis with the rather characteristic history of tracheobronchitis in the absence of physical signs and roentgenographic evidence of disease should strongly suggest this condition. If the symptoms are persistent, bronchoscopic examination should be employed for diagnostic and therapeutic purposes.

The many less frequent causes of hemoptysis are to be considered after the above have been ruled out by exhaustive diagnostic studies and the further differential diagnosis must depend upon the process of elimination. In 40 years of private practice Ware [2] observed 386 cases of hemoptysis. In his interesting report it appears that 68 of the cases were followed from two to 37 years without subsequent symptoms of pulmonary disease. It must be remembered that these cases were reported in 1860 and did not come under the sharp scrutiny of modern methods. Blood spitting which can not be explained after searching diagnostic examinations, including all roentgenographic and laboratory studies and bronchoscopy, demands continued observation and repeated examinations until a diagnosis is arrived at or a definite interval of safety has passed.

The emergency treatment of hemoptysis presents a fertile field for the ancient art of medicine. Very few patients escape serious psychological conflicts when they witness the issue of blood from their own throats. Immediately, they anticipate death, advancing disease or both. If emotionally unstable, they are panic stricken; if well poised, they may be no less concerned, but through cultivated inhibitions they may conceal serious psychological conflicts. Since sedation is the most important immediate therapeutic indication, it is wise to remember that psychological sedation through artful reassurance is much safer than soporific sedation which deadens the cough reflex and endangers the patient's future. Judicious, authoritative command of the agitated patient and the distraught relatives prepares the way for scientific management according to individual needs. Often the above plan must be supplemented by mild sedative medication. The hypodermic use of codein or morphine in small doses may be indicated. In many cases bromides or barbiturates by mouth may be sufficient. If the psychological approach has been successful, cough and physical agitation may be brought under voluntary control, thus limiting the questionable employment of the more powerful sedatives. In obstinate cases, however, morphine may prove to be the splint necessary to temporarily control the break. We must admit that laudanum was the friend of nearly all the old time "lungers" who did not have the benefit of modern therapy.

After securing the maximum mental and physical rest, other therapeutic considerations arise. We may dismiss all the medicinal agents designed to increase the coagulation of the blood by the admission that aside from their psychological effects, they are virtually useless unless blood tests demonstrate a definite need for them.

Absolute rest in bed is essential. If hemorrhage is profuse and the expectoration of blood is not satisfactory, a favorable posture for drainage should be sought. This is doubly important if sedatives have impaired the cough reflex. Mechanical measures have assumed a position of increasing importance, both in the emergency and in the treatment of continuous or recurrent hemoptysis. Time will not permit a detailed account of these measures, but attention is called to the fact that they range from simple procedures to restrict pulmonary expansion and undue respiratory agitation when the patient is seized with paroxysms of coughing, to collapse therapy which includes artificial pneumothorax, phrenic nerve interruption, thoracoplasty, lobectomy and pneumonectomy.

Long before the author was fully apprised of the importance of mechanical influences in the control of hemoptysis, a ranchman was admitted to the Farm Sanatorium because of recurrent profuse pulmonary hemorrhages. The history indicated that when it seemed that death might conclude the initial attack, two of the boys on the ranch tied a rope around his chest as they would tie the lariat of a bucking broncho around a snubbing post. Apparently strapping the thorax down tightly controlled an attack which occurred while he was in the Sanatorium. Since then, the author has often employed strapping or a tight bandage over the lower half of the thorax and the abdomen with good mechanical and psychological effect. When all the less radical procedures fail or if the emergency is too urgent for such deliberate therapy, artificial pneumothorax should be given a trial. As a rule, with the aid of the patient, who often has a definite sense of location, the clinician can determine which side is bleeding. If this is impossible and roentgenographic studies are not feasible or available the best possible guess should be considered a sufficient guide for the institution of pneumothorax. Usually such a guess is adequate for control, provided adhesions do not render collapse impossible or unsatisfactory. It is possible the altered intrathoracic pressure may exercise a favorable influence even though bleeding is not coming from the collapsed lung. Recently in an emergency, the author after hurried auscultation of the chest, collapsed the left lung for the control of alarming recurrent hemoptysis. He was guided by extensive bubbling râles over the left lung. Though the bleeding was promptly controlled, later diagnostic studies, including roentgenograms, suggested the right lung as the most probable source of bleeding. On two occasions simultaneous bilateral pneumothorax has been considered necessary to control persistent dangerous hemorrhage.

If artificial pneumothorax is not successful, phrenic nerve interruption should be considered. The successful use of pneumoperitoneum combined

with interruption of the phrenic nerve has been reported. When all other methods fail, thoracoplasty may be considered for the control of recurrent hemoptysis. In neoplasms, bronchiectasis, and occasionally in lung abscess and cystic disease, lobectomy or pneumonectomy may be indicated.

Summary

Brief consideration has been given to the development of our knowledge of hemoptysis; the various sources of hemoptysis and the relative importance of the principal causes; the differential diagnosis; the treatment, in which reassurance and other psychological measures are stressed as being more important than promiscuous dosing with sedatives and coagulants; and finally, mechanical agencies which in some cases are life saving.

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DIRECT MEASUREMENTS OF THE EFFECTS OF BROMIDES, SODIUM AMYTAL AND OF CAFFEINE IN MAN*

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MANY publications appear each year concerning the influence of stimulants and sedatives in animals and in man. Investigations have afforded much indirect evidence of the action of such agents on the nervous and muscular systems. However, direct quantitative measurements of functional states in the intact organism following the administration of pharmacological substances have never been attempted, doubtless because of lack of suitable instruments. The possibility of such investigation has finally been opened by advances in electrophysiology in the last decade. Most convenient for the present investigation, which I believe marks a first step in this special field, is apparatus capable of registering the average microvoltage, yielding values which can be plotted against time, as recently described.[1] Comparison of the data secured under controlled conditions, before and after such administration, can lead to knowledge of considerable practical as well as scientific importance.

Since no method has been developed to estimate the activity of the intact nervous system as a whole at any instant, we continue to be limited to sampling methods applied to a particular section of the organism. For approximately one half century, the prevailing custom among those who wished to test the functional condition of the nervous system was to determine the excitability of the knee-jerk and other deep reflexes during the resting state.[2] An alternative procedure would be to measure action-potentials against time in the quadriceps femoris muscle, as reported recently.[3] Likewise, variations in contraction in the flexors of the forearm can be conveniently studied under controlled conditions, when the arm is supported in a position of rest.[4] In line with the traditional view deriving as above mentioned, the results of recordings on more than 300 individuals during the last decade seem to justify the belief that the electrical state of the right arm flexor muscles can afford a sample of considerable value toward indicating the functional state of the organism at any instant and, therefore, can serve as a therapeutic test. That the effects of motor stimulants and sedatives are registrable in neuromuscular responses is not surprising, for this must evidently be possible if they act upon motor centers, anterior horn cells, peripheral nerves or on muscles directly; but even if they act directly only on higher centers in the brain, a consequent increased or decreased response there will determine increase or decrease of efferent discharge as a secondary but inevitable effect.

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The present investigation concerns the influence of bromides, sodium amytal and caffeine on man, as far as can be determined in terms of action-potentials.* Many contributions have followed the invention of concentric electrodes suitable for recording potentials from single fiber groups by Adrian and Bronk.[5] However, as stated previously: "In seeking a quantitative indication of tonus in a muscle as a whole, we are led away from the employment of micro-electrodes and of concentric electrodes, which were found suitable in the investigation of the electrical activity of single fibers or of small groups of fibers discharging as a unit. If it were possible to place a pair of concentric electrodes in juxtaposition with each and every muscle fiber in the muscle group tested and so to measure each and every discharge, a plot of voltages and frequencies against time for all the fibers would be an ideal electrical measurement of contraction in the muscle group. Since this is beyond possibility, wire electrodes are inserted into the muscle tissue where an indefinite number of fibers discharge variously from instant to instant. Obviously, at any instant the p.d. in the electrodes is the resultant from discharges in the surrounding muscles mass." [3] Accordingly the method presently employed depends upon the number of motor units in action and the frequency of discharge in each motor unit.

As one of the many antecedents to the present study, it was necessary to determine whether lesser grades of contraction, including what is commonly called "tonus," are likewise marked by correspondingly smaller electrical changes. For this purpose, apparatus capable of yielding measurements of transient voltages lower than had previously been employed in medicine and in most departments of physics was accordingly developed. It was found that the slightest degrees and variations of muscular contraction, including tonus, could be electrically measured, even in the intact animal, if the voltage sensitivity was sufficiently high.

When the string galvanometer is employed, as in electrocardiography, but with special amplifier equipment, the photographed deflection of the string can be made as great as one centimeter per microvolt—which is about one thousandfold the magnification seen in heart records. Obviously, when amplification of such high order is used, it is necessary in each study to make control tests in which suitable resistances ("inanimate objects") replace the living tissues under investigation. Such control tests afford values which must be allowed for in order to arrive at final quantitative determinations.

Measurement of photographed string deflections occurring at various rates per second involved great labor when a technician had to examine each line individually with the aid of a magnifying glass. Accordingly, newer equipment was devised whereby much of the labor of measurement is performed in the instrument itself. With the Integrating Myovoltmeter (or Neurovoltmeter), as the new instrument is called, action-potentials (within certain limits) are measured without photography. The instrument includes

* Ampules of sodium amytal were kindly furnished by Eli Lilly & Company; caffeine by Abbott & Company.

an amplifier of standard type, with a characteristic fairly flat for frequencies from 20 to 10,000. The amplified potentials are rectified and averaged over each two minute period during the test. These averaged values can be plotted against time, representing the results of the test. To standardize the instrument, 1 microvolt a.c. at 57 cycles is applied across the input terminals of the amplifier and readings are made on the galvanometer dial for two minute periods. From the mean of several such readings is subtracted the mean determined from the same number of readings made through the same circuits under identical conditions except that no current passed through the standardizer. The mean value thus determined per microvolt per two minute interval is divided by 2, in order that all results in the present investigation can be stated in terms of microvolts d.c.

At any instant the total rectified output is indicated by the position of a needle on the dial of a microammeter. Fluctuations of this needle enable the investigator visually to follow the variations in contraction in the muscle studied; if the subject extends his leg, the needle moves up on the dial accordingly and stays up as long as the extension is maintained; but if the muscle becomes limp and relaxed, the needle turns toward zero.

Since the potentials to be considered demonstrably occur upon muscular contraction, whether during movement or the maintenance of a steady state, I shall hereinafter use the term "integrated action-potentials" for what are customarily called "action-currents" or "action-potentials."

Electrodes consisted of platinum iridium wires about 11 mm. long and 0.011 inch in diameter inserted perpendicularly into the tissues. As a rule these were inserted crosswise in the right biceps-brachial muscles about two inches above the apex of the angle formed by the skin when the forearm is bent to about 90°. They were about 2½ inches apart and were equidistant from the volar midline. This location minimizes the electrocardiogram which tends to appear when electrodes are located lengthwise in these muscles.

Fifteen subjects were employed without advance information as to the purpose of the study. They were adults of both sexes actively engaged in business pursuits of various kinds * and were as a rule in fair health, as found upon examining them physically. Although some were of nervous disposition, as a group they seemed fairly representative of average or "normal" individuals. Every effort was made to prevent them from learning the nature of the medicine administered. This effort appeared to be successful except in one or two instances, when the color of the triple bromide solution betrayed its character to the subject who was familiar with it.

During the tests the subject lay in a semi-soundproof room. Conversation was not permitted. The instruction was to lie quietly with eyes closed. A few minutes were permitted for him to "settle down" in each instance, before recording was initiated. Previous experience with similar tests without medication suggested that a 30 minute period of recording would prob-

* There were three tailors, three waiters, two office girls, two vault clerks, one elevator operator, one plumber, one mailman, one medical secretary and one clerical manager.

TABLE I

The values represent contraction-voltages for each subject with and without medication (averaged for three periods following each type of medication or for three corresponding control tests).

Subject	Sex	Age	Without Bromides	With Bromides	Sodium Amytal	Sterile Water	Caffeine
1	F	22	1.58	1.02	.73	1.12	1.26
2	F	21	.49	.38	.27	.34	.35
3	M	47	2.04	1.05	.67	3.54	4.76
4	M	54	.67	.18	.21	.24	.84
5	F	34	.20	.26	.08	.76	.86
6	M	26	.24	.22	.26	.25	.56
7	M	42	.56	.37	.34	.23	1.03
8	M	40	3.21	2.44	1.02	1.85	2.02
9	F	31	2.65	1.66	.68	1.42	2.90
10	M	44	.39	.53	.42	.31	.67
11	M	42	.70	1.04	.61	.38	.95
12	M	51	.28	.21	.33	.38	1.04
13	M	44	.49	.59	.19	.54	.46
14	M	63	1.61	.42	.24	.28	.38
15	M	43	1.33	.42	.31	.83	1.83

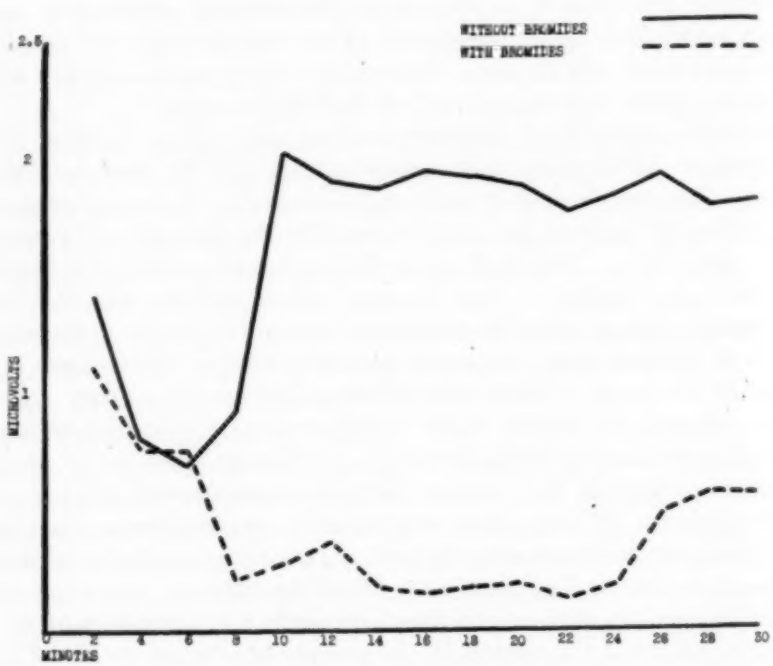

FIG. 1. After the administration of 90 grains of triple bromides by mouth, the curve for subject 14 shows a measured decrease of electrical activity (broken line), as compared with similar tests without medication (unbroken line).

In this and in figures 2, 3, 6 and 7, each curve represents a composite of three 30 minute tests made on different days. In all figures each point is plotted to indicate the micro-voltage averaged for the preceding two minutes.

ably prove sufficiently long for present purposes. No attempt was made to determine the minimum effective dosage, but it was desired if possible to employ with all subjects a uniform dose which might have an observable result in most if not in all instances. Therefore, in a trial experiment with one subject, 30 grains were given by mouth (twice the dose of triple bromides standardized in the National Formulary) but (contrary to the custom in

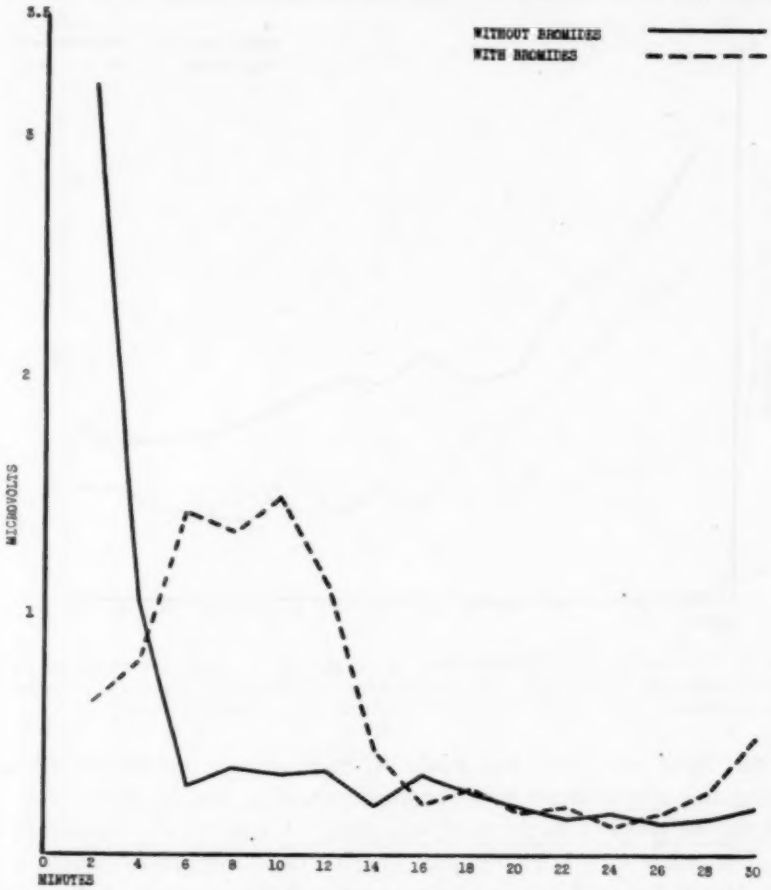

FIG. 2. Subsequent to the administration of 90 grains of triple bromides by mouth, the curve for subject 13 shows measured increase of electrical activity (broken line) after the first four minutes, as compared with similar tests without medication (unbroken line). This increase persists for about 10 minutes, after which the curves show little difference. In this test, no indication of sedation is manifested.

clinical practice) the subject was kept in ignorance as to the nature of the medication. Upon inquiry, he stated that he failed to notice any difference in sensations or in any other respect as compared with times when he had taken no medicine. Accordingly, it was decided to administer six times the standard clinical dosage by mouth, namely 90 grains, half two hours before the test and half one hour before the test.

When this was done contraction-potentials were lowered moderately in most instances as compared with control tests made on different days, in which no medication at all was given, but in which every other procedure and condition present on test days was duplicated as far as possible. Subjective effects were less marked than would generally be anticipated; two subjects complained of a little dizziness; several stated that, judging by their feelings, they would not have known that they had taken any medicine at all.

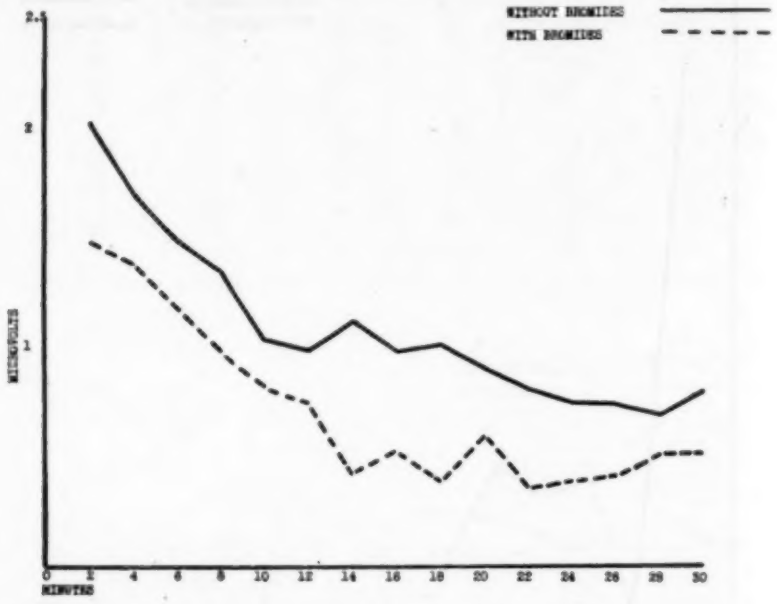

FIG. 3. Following the administration of 90 grains of triple bromides by mouth, the composite graph for 15 subjects shows a measured decrease of electrical activity (broken line), as compared with similar tests without medication (unbroken line).

A half-hour recording was made on each subject on three different days following the administration of bromides, and a control test under similar conditions but without medication was made on each of the three intervening days. For each half-hour of test, the average integrated action-potential value is found for each two-minute period and the average of these averages is then calculated for the three tests, both with and without bromides (table 1, columns 4 and 5). Comparing these values, a decline followed the administration of bromides in 11 of the 15 instances, but an increase in the four others. (In two of the instances of decline as well as in two of the instances of increase, the change was less than 10 per cent.)

The curve for the most marked instance of decline for any subject is shown in figure 1,* and one showing an increase for about one-third of the

* Subjects requested to sit quietly for a prolonged period often exhibit a rise of integrated action-potentials after the lapse of a variable interval. They report that they become impatient and desire to move about. This might explain the rise of the unbroken line

period and an approximate equality for most of the remainder appears in figure 2. Evidently even following six times the standard dosage of bromides, the effect can be manifestly negative in exceptional instances under the conditions studied.

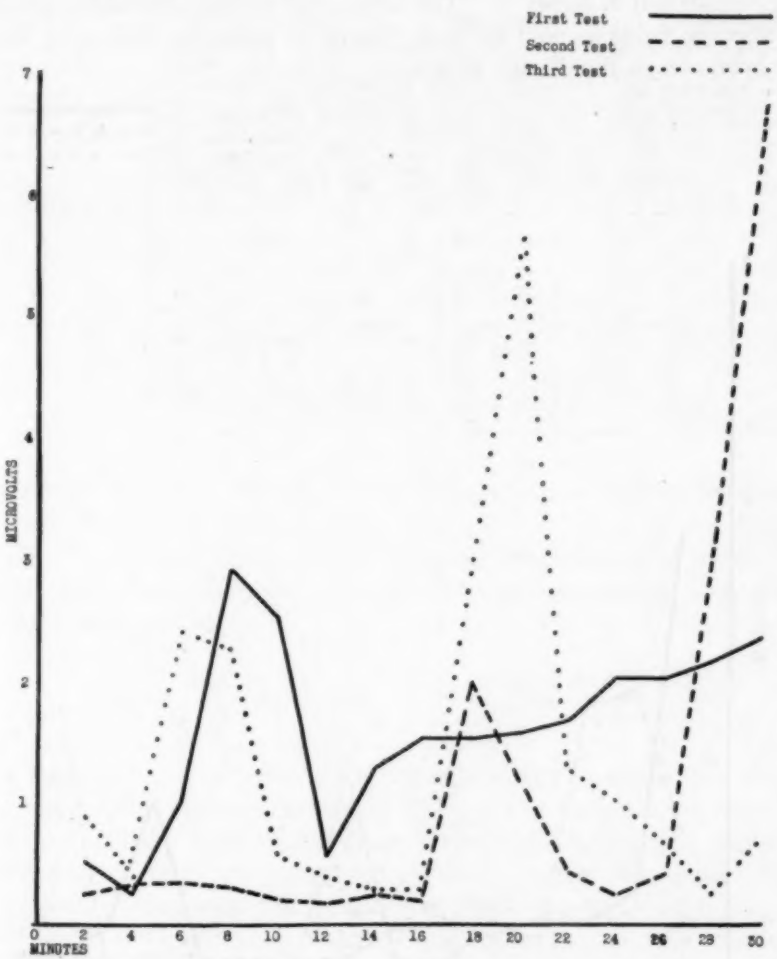

FIG. 4. These curves for subject 15 (plumber) illustrate the character and magnitude of daily variations in control tests (when no medicine was administered).

However, if the results for all subjects are included in a composite graph, a distinct if moderate effect of the bromides is indicated (figure 3). Integrated potentials in the muscles tested are (on the whole) lowered during the period investigated.

The subjects show considerable daily variation in the unmedicated con-

at 10 minutes and thereafter seen in figure 1. The effect of the sedative might be to nullify this tendency toward increased tension, indicated by increased action-potentials, so that it fails to appear in the curve following medication.

dition, but when the results are positive the tendency of the sedative seems to smooth out the curve somewhat. This is illustrated in one subject by the individual curves for three different days without bromides shown in figure 4, in comparison with those for three intervening days on which bromides were given shown in figure 5. The integrated action-potential is diminished in its average value as well as in its degree of variation following ingestion of the bromides in the dosage stated.

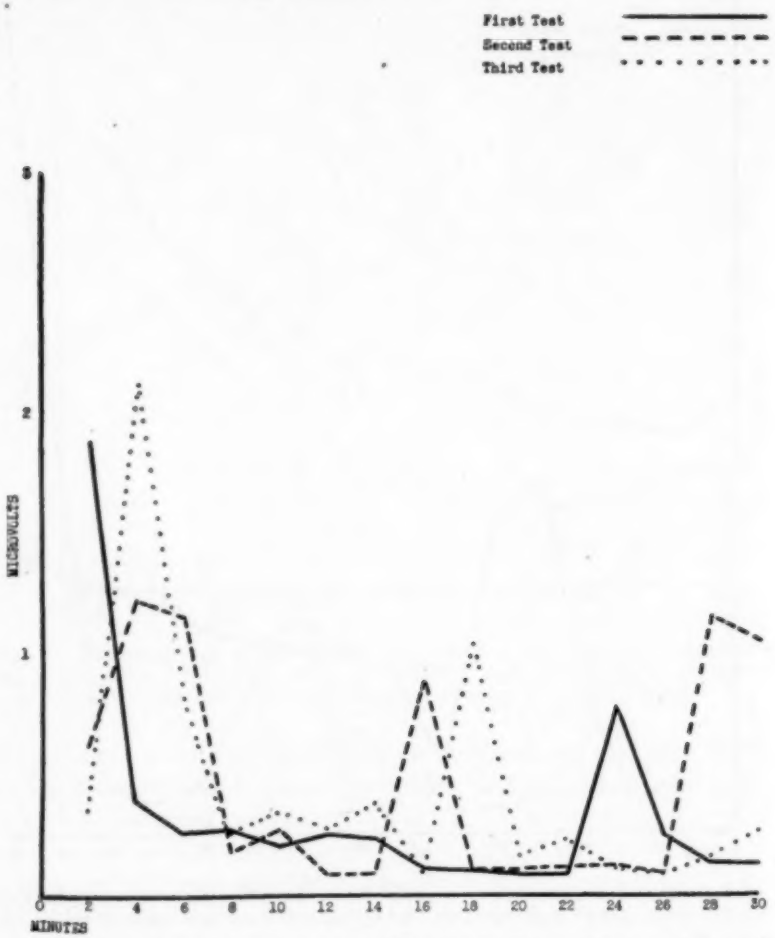

FIG. 5. In these curves for the same subject as in figure 4 but on intervening days, following the ingestion of bromides, the integrated action-potential averages less and the daily variation is diminished.

At the beginning of many of the observation periods, a relatively large drop in integrated potential occurs. This presumably marks the individual's becoming more relaxed as he adapts himself to the conditions of test, as discussed previously. In some instances (as in the control test in figure 1), the adaptation proves shortlasting.

TABLE II

Results in subject 15. Figures in the first two rows indicate the range of microvoltage variation in each test. Those in the third row show the mean values of the determinations for tests indicated by the headings of the columns. Variance and probable errors appear in the fourth and fifth rows.

		Without Bromide				With Bromide				Sterile Water			
		Test No. 1	Test No. 2	Test No. 3	Total	Test No. 1	Test No. 2	Test No. 3	Total	Test No. 1	Test No. 2	Test No. 3	Total
Range	High	2.92	6.77	5.66		1.88	1.24	2.12		4.32	.49	4.10	
	Low	.24	.16	.24		.09	.09	.10		.11	.09	.13	
Mean		1.59	1.08	1.32	1.33	.34	.48	.45	.42	.97	.16	1.36	.83
		.77	1.76	1.57	.91	.46	.47	.54	.32	1.33	.11	1.33	.68
P.E.		.134	.306	.173	.158	.080	.082	.094	.056	.231	.019	.231	.118
		Caffeine				Sodium Amytal							
		Test No. 1	Test No. 2	Test No. 3	Total	Test No. 1	Test No. 2	Test No. 3	Total				
Range	High	3.47	6.92	5.00		1.86	.81	1.73					
	Low	.27	.30	.45		0	.03	.11					
Mean		1.36	2.23	1.89	1.83	.22	.24	.47	.31				
		1.006	1.84	1.64	1.29	.46	.17	.47	.29				
P.E.		.175	.320	.285	.224	.080	.030	.082	.050				

The range of daily variation is illustrated also by the data secured for one subject (No. 15) as shown in table 2 (rows 1 and 2). In the third row appear the mean values, while the variance and probable error of the mean appear in the remaining rows. These values are calculated according to standard formulae, namely,

$$\sigma X = \frac{\Sigma(X - \bar{X})}{N - 1} \quad \text{and} \quad \text{P.E. of } \bar{X} = \frac{.6745X}{\sqrt{N}}$$

For this subject the total value with bromide (.42) is much less than the corresponding value without bromide (1.33). The reduction is more than five times the probable error of the mean, a result which is clearly significant.

Another set of tests was conducted similarly, except that sodium amytal was administered intramuscularly and the effect measured beginning one-half hour later. The dosage, grains 1⅞ was selected as unlikely to interfere with the subjects in their daily work. However, subjective effects such as sleepiness seemed considerably greater than those following the bromides. We can compare the averaged values for the barbiturate (determined as stated above) with those determined on intervening days, when sterile water was injected, assuming that our control conditions should as far as possible duplicate the tested conditions. To this end the tests following bromides administered by mouth were generally alternated on successive days with tests without medication, and the tests following injections were also grouped together in point of time as far as possible. It is possible that the injection of a solution has psychological effects which should be considered. There-

fore, we assume that the results following the injection of barbiturate and of caffeine are comparable with the injection of sterile water, rather than with those labeled "Without Bromides." Otherwise, admittedly, if the sterile water figures are taken as a control, the effect of the bromide appears not to be significant.

Comparing the results for the 15 subjects following the administration of sodium amytal as stated above, 13 showed declines, but four of the latter

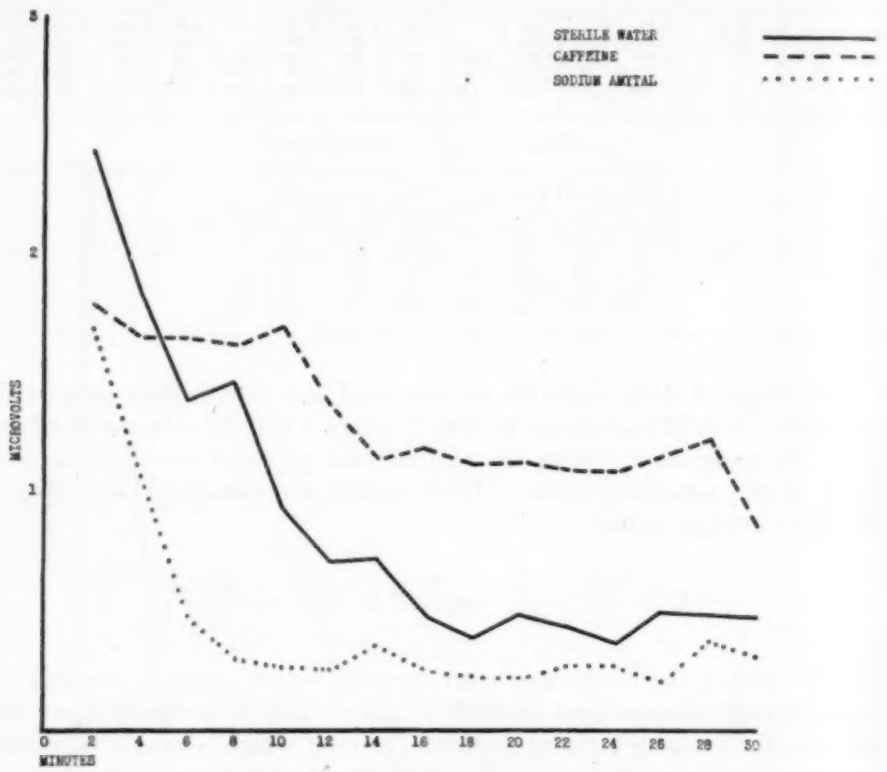

FIG. 6. Following the intramuscular injection of 1⅞ grains sodium amytal, the composite graph for 15 subjects shows a measured decrease of electrical activity (dotted line) as compared with the results following a similar injection of sterile water (unbroken line). Following the intramuscular injection of 7½ grains caffeine sodium benzoate, the graph (broken line) shows a measured increase of electrical activity, as compared with the control (unbroken line).

were below 10 per cent. The other two subjects showed increases exceeding 10 per cent. These results are revealed in the averages shown in table 1, column 6, which can be compared with the averages for the controls (sterile water) shown in column 7.

If the results for all the subjects are included in a composite graph, a distinct decline in contraction-potentials is obvious, as compared with the graph for the control tests following injections of sterile water (figure 6).

The composite graph following the intramuscular administration of sodium amytal (gr. 1⅞) indicates distinctly lower contraction-potentials than that following the oral administration of the bromides (gr. 90).

In subjects whose control tests show little tonicity, the reduction effected by the barbiturate may reduce the contraction practically to zero level. This is illustrated in figure 7, where the composite values for subject 5 are shown, following the injection of sodium amytal as well as of sterile water. We

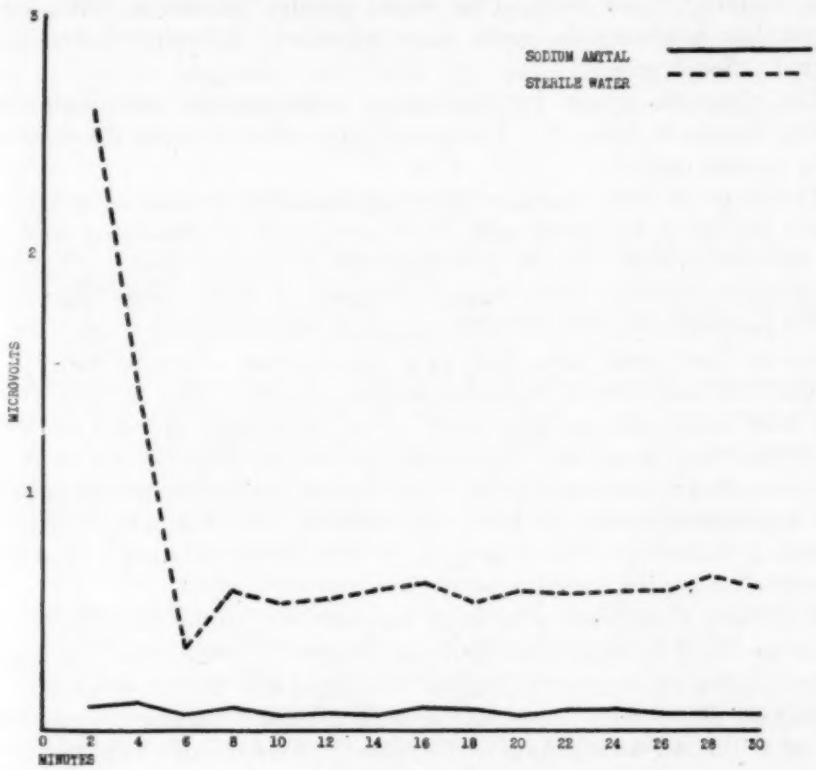

FIG. 7. Following the intramuscular injection of 1⅞ grains sodium amytal, the composite graph for three tests for subject 5 shows a measured decrease of electrical activity approximately to the zero level (unbroken line), as compared with the results following the injection of sterile water (broken line). Conditions as in figure 1.

can assume that probable errors of measurement due to the instrument itself amount to 0.01 microvolt. Accordingly, the tonus level for this subject following the administration of the barbiturate, appears to be approximately but not quite at zero.*

Caffeine sodium benzoate (gr. 7½) was injected intramuscularly one-half hour before the tests. Few subjective effects were noted. The subjects

* When the mean of 10 readings, for example, was 17.3 scale divisions per microvolt a.c. impressed upon the amplifier, the probable error of the mean was .15, which is less than 1 per cent.

did not fall asleep as often following the administration of this stimulant as they did following that of the bromides and particularly that of the barbiturate. At least one, however, fell asleep during the test (subject 12 in table 1).

A marked increase of contraction-potentials following the administration of caffeine (as compared with that of sterile water) is noted for most subjects. Of the 15 subjects, 14 show an increase and for all but one the increase exceeds 10 per cent. One shows greater relaxation following the caffeine than following the sterile water injection. All subjects were mostly habitual coffee drinkers.

The composite graph for the results following the administration of caffeine appears in figure 6. The stimulating action is evidently measurable by the present method.

The range of daily variation following the administration of caffeine and sodium amytal as compared with sterile water can be illustrated once more with one subject (No. 15), as shown in rows 1 and 2 of table 2. Under the corresponding headings in the third row appear the mean values; the variance and the probable error of the mean appear in the remaining rows. For this subject the total mean value following the injection of sterile water is .83, and that following the injection of caffeine is 1.83. The increase is more than four times the probable error. Following the injection of sodium amytal the total mean value is reduced to .31, a reduction which is more than four times the probable error. The figures can be interpreted as indicating a significant increase in the contraction-potentials following caffeine and a decrease following sodium amytal, in both instances compared with the control following the injection of sterile water.

Subjective complaints were most frequent concerning the effects of the sterile water. One individual (subject 7, table 1) stated that the injection led to fainting spells such as he had not experienced since boyhood. Apparently in all subjects there was a tendency for "burning" sensations to develop at the site of injection of the water. Perhaps this might have been averted if physiological saline solution had been employed instead.

Discussion

Quantitative measurements can be applied to the nervous system directly. For this purpose, an electrode is inserted into any nerve at some locality near the surface, for example the ulnar nerve in the fossa adjacent to the olecranon process. This procedure has been outlined previously.[6] Following my experience with these methods, there is no reason to assume that results so secured would differ in the gross from those presented herein, but extension of the investigation in this direction is indicated.

It seems irrelevant to review here countless previous investigations on the effects of stimulants and sedatives, whatever their importance, inasmuch as the results have been chiefly in terms of reactions of the organism or of

changed bodily chemistry. Changes in the electroencephalogram following the administration of drugs have engaged the attention of various investigators since the time of Berger.[7] Stimulants such as caffeine may or may not produce a direct, registrable effect on brain waves, but sedatives will do so, provided they induce a somnolent state (Berger). "Sedatives cause changes similar to those observed in normal sleep" (Gibbs et al.). However, the qualitative variations between individuals are considerable,[8] the source of the waves not yet certain,[9] and the effects of drugs provide additional variations affording little hope of securing data of quantitative significance. No one up to date has suggested that brain wave recordings yield data suitable for the quantitative registration of the influence of stimulants or sedatives on the nervous system.

Summary and Conclusions

1. Direct measurements can be obtained of the effects of stimulants and sedatives (and other types of medication) on the muscular and nervous systems in terms of action-potentials, if suitable electrodes are placed in neuromuscular regions.

2. Measurements should be accurate to a very small percentage of a microvolt d.c. within a selected frequency range. For quantitative purposes, the Integrating Neurovoltmeter (or Myovoltmeter) is most convenient. Potentials in muscle during a period of test can be averaged for unit periods and plotted in microvolts d.c. against time. In the present investigation this was done for the flexor muscles of one arm in 15 subjects in a fair state of health. The data apparently can afford some index of the influence of stimulants and sedatives on the effector portion of the nervous system.

3. Bromides by mouth in customary dosage (15 grains) would appear to have little if any measurable effect on some normal individuals according to the present test. This suggests that therapeutic results from such small doses probably often depend upon the knowledge of the patient that he has taken a sedative.

4. Bromides in large dosage (90 grains within two hours) affected some of the individuals tested but little, if at all. However, according to the subjective reports as well as the objective findings, the composite graph for the 15 subjects shows diminished potentials as compared with controls when no medication was given.

5. A greater quieting effect on the neuromuscular system was notable in the composite results following the intramuscular administration of sodium amytal (grains 1⅞).

6. Caffeine sodium benzoate (grains 7½ intramuscularly) produced little effect in some instances, but a definitely stimulating effect was noted in the composite graph, as compared with the results following the injection of sterile water.

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SOME CLINICAL CHARACTERISTICS OF MUMPS, AND THE EFFECT OF BELLADONNA IN TREATMENT; A STUDY MADE AT THE STATION HOSPITAL, FORT GEORGE G. MEADE, MARYLAND *

By HAROLD W. POTTER, F.A.C.P., LT. COL., MEDICAL CORPS, ORC, and LEWIS H. BRONSTEIN, CAPT., MEDICAL CORPS, A. U. S.

IN civilian practice mumps is considered a disease of children and, therefore, is not considered much of a problem from the standpoint of disturbing the life of a community. The adults who contract the disease are usually the butt of jokes of their friends and are infrequent enough to cause comment only from that standpoint.

In military life, however, mumps may become much more of a problem, not from a disabling standpoint, but from the fact that it might fill hospital beds which are needed for other types of sick patients. The length of time the soldier has to stay in the hospital also causes interference with the training program that his organization is undertaking.

The study of the following consecutive 124 cases was originally undertaken in an attempt to ascertain the value of treating such patients with belladonna. The use of this drug had been suggested to shorten the course of the disease and to decrease the incidence of complications. The study of these cases revealed some facts at variance with previous recorded material in the literature, as well as confirmation of other statements. Future studies of more cases may either confirm or discount these observations. However, it is hoped that this will give impetus to more observation in a disease which has been accepted in most quarters as "just one of those things."

Excellent reviews of this disease [1, 2] are present in recent literature. It is deemed unnecessary, therefore, to discuss all the aspects of the condition.

All of these cases were not suitable for every type of analysis. In some instances, for example, the exact date of determination of the swelling could not be determined from the clinical record. In determining the incidence of orchitis, the members of the W.A.A.C. who had mumps were excluded because none of them had oöphoritis and because oöphoritis is a rarer complication than orchitis.

Effect of Belladonna. Forty-two consecutive cases of mumps were treated with belladonna and were compared with the previous 82 cases which were not so treated. The untreated cases were seen from October 1942 through March 1943 and the treated ones from March 1943 through May 1943.

* Received for publication August 16, 1943.

A preliminary analysis of the 82 untreated cases showed no significant difference, as far as duration of disease was concerned, in those patients who entered with a unilateral lesion which did not spread, with a unilateral lesion which became bilateral, or with a bilateral lesion. The comparison was, therefore, made only in regard to the day of the disease on which the soldier entered the hospital. There were too few cases in the belladonna series who entered on the third day or subsequent day for adequate comparison of duration of illness. Accordingly, only the series for the first and second day were compared.

The medication was administered as follows: On admission, the soldier was given gr. 1/100 of atropine sulfate and then 1 c.c. of the tincture of belladonna by mouth every two hours, until atropinization, as evidenced by dilatation of the pupil and/or dryness of the throat, was produced. Subsequent medication was given in doses to keep these changes constant, either until the patient was cured or until gastrointestinal discomfort made it necessary to discontinue the drug. The 1 c.c. was measured out by hypodermic syringe in order to have as accurate a dose as possible.

The patients who received belladonna had as much discomfort from their parotitis as did those who did not receive the drug. In addition, some had abdominal distress from the high dosage of drug that had to be given.

TABLE I

Duration of Illness—Treated with Belladonna and Untreated

	No. of Cases	Mean No. of Days	Standard Deviation	Probable Error
A. Admitted to hospital on first day of illness				
Untreated	24	11.71	3.3	0.46
Treated	11	11.00	3.69	0.62
B. Admitted to hospital on second day				
Untreated	33	13.3	4.18	0.491
Treated	16	12.8	2.76	0.165

A study of table 1 will show that there is no statistical significance to the difference between the durations of the disease in these groups.

In each series of cases there was one case of meningo-encephalitis. Table 2 A shows the incidence of orchitis in the two series. In this grouping, the females were excluded as were those patients who entered the hospital with orchitis. A comparison of the percentages by means of either the standard or probable error method shows that no statistical significance can be attached to the difference between them.

Table 2 B shows the average duration of the orchitis if we again include those patients who entered with this complaint. Again no significance can be attached to this difference.

TABLE II

	Total No. of Cases	No. of Cases Developing Orchitis	Percentage Incidence
A. Development of Orchitis			
Untreated	77	7	9
Treated	35	6	17.1

B. Duration of Orchitis*	No. of Cases	Mean Duration
Untreated	11	8.7
Treated	8	7.0

* In one case who entered with orchitis and parotitis, the day of termination of illness could not be determined.

It is thus apparent that belladonna given as indicated above had had no effect in cases of mumps on the following events in the course of the disease: (1) duration of illness; (2) incidence of complications; (3) duration of orchitis; (4) comfort and well being of the patient.

Origin and Race of the Soldier. The largest series of cases to be published from studies in young adults was from World War I.[3] The statement was made that most of the soldiers came from rural communities. This statement has been repeated time and time again and has been quoted as late as 1940.[1, 2] We, therefore, reviewed our cases from that standpoint.

It is rather difficult to decide where to place the boundary between rural and urban communities. We used the home address of the soldier as well as his civilian occupation in an attempt to decide in which category he should be placed. It was decided that a town's population of 10,000 would be a dividing line. It is realized that this probably helps throw the weight of the figures to the rural group. In addition, many soldiers who had migrated to a city to work have no permanent address there and are more likely to give their parents' address which might be in a small community. It is with this knowledge that the results are more unusual in that 50 soldiers came from urban areas and 72 came from rural areas. One soldier gave his address as no home and information was lacking in another.

The cases were broken down to determine the race of the soldier and his origin. Table 2 shows that there were twice as many white as colored soldiers admitted with mumps. However, the population of the camp had a much greater proportion of white to colored soldiers during this time so that there is a greater incidence among the colored soldiers, as has been noted previously. From the same table, it can also be seen that the colored soldiers had a preponderance of rural origin as compared to the white soldiers. The greatest majority of these men came from the Southern states.

These figures indicate that in our series there was not the great difference noted previously between rural and urban groups. This difference is gradually being destroyed with our changes in civilization, such as improved methods of transportation, consolidated school systems in rural areas, etc., which make for greater contact among people. The group which is still isolated to the greater degree from the standpoint mentioned above are the negroes of the South. The figures show that some distinction between rural and urban populations holds. However, it is not as marked as previously recorded. Should analysis of a larger series of cases bear out this finding, a change in our concept should be recorded in textbooks and literature.

Duration of Service. With an ever-expanding Army, there is a continuous influx of new recruits into the service so that communicable disease should always find a fertile field in which to spread. Wheelis,[4] in considering this factor for communicable disease generally, found that the highest rates occurred during the first two months of military service. However, he advises study for individual disease. Ravdin,[3] in discussing the mumps cases at Camp Wheeler during World War I, states that the length of service in 95 per cent of the cases was two months. Our cases were, therefore, considered from this standpoint.

In analyzing the figures it became apparent that there were two ways of viewing them. It was possible merely to take the length of the soldier's service in the Army at the time he developed his mumps. Table 3 shows

TABLE III*

Color and Origin

Total No. of Whites	Rural	Urban
80	42	37
43	30	13
Colored		
43	30	13

* One case record not complete. One white patient had no address.

this in column 1 in relation to intervals of two months' service. However, such a table would not take into account those soldiers who entered during the summer when mumps was not prevalent. Our cases ran from October through May. There were only two cases during the last week of October. The cases really began to come in in November. That month was, therefore, chosen as the first month of the mumps season and the duration of service was related to November 1942. Thus, a soldier who came down with mumps in January 1943 and had six months' service was credited as having only three months' service. Obviously, those soldiers who had one or more years' service were credited with their full time. This correction is seen in column 3. The appropriate percentages can be found in columns 2 and 4.

These figures show that our series of cases do not follow the expected relationship as recorded in the literature. The uncorrected series shows that 26.66 per cent of the cases had only four months' service and 39.46 per cent one half year's service. The largest groups were those with eight and 10 months' service. The corrected series shows that 41.66 per cent of the cases had four months' service and 58.32 per cent had up to six months' service. In both groups, approximately 85 per cent had their mumps within the first year.

It would seem that the time limit for developing mumps in susceptibles should be extended from two months to one year in order to include all of them. This longer exposure may be due to a slightly acquired resistance with greater urbanization in the years following World War I. This would fit in with the previous facts that urban or rural distribution has changed. However, more cases are necessary to verify this.

Duration of Disease. It was thought that the day of hospitalization and therefore beginning of treatment would have some effect on the duration of the disease. Our treatment, including those who were tested with belladonna, consisted of regular diet, mouth wash where indicated, and absolute bed rest, which was not always observed by the soldier.

TABLE IV*

Relation between Onset of Mumps and Duration of Service

	I Uncorrected	II Percentage	III Corrected to November 1942	IV Percentage
Up to 0–2 months	16	13.33	25	20.83
Up to 4	16	13.33	25	20.83
6	15	12.50	20	16.66
8	26	21.66	13	10.83
10	22	18.33	15	12.50
12	7	5.83	5	4.16
14	2	1.66	2	1.66
16				
18	1	0.83	1	0.83
20	1	0.83	1	0.83
22	3	2.50	3	2.50
24	5	4.16	5	4.16
26				
28	3	3.50	3	2.50
1–3 yrs.	2	1.66	2	1.66
4 yrs.	1	0.83	1	0.83

* Four cases omitted—one case, length of service was not recorded. Three cases were admitted to hospital on first day in Army with mumps having been present for four, five, and nine days.

Table 5 was set up with this in mind. It shows that there is no significant difference if the cases that entered the hospital on the first, second, and third days are considered. Those who entered on subsequent days were too few in number to help from a statistical standpoint. The known num-

TABLE V

Relation of Day of Hospitalization to Duration of Disease

	No. of Cases	Mean	Standard Deviation	Probable Error
1st Day	41	11.4	3.57	0.37
2nd Day	49	13.1	3.67	0.353
3rd Day	12	13.5	2.94	0.6

ber of days is the total number that the soldier had his disease. If only the days of hospitalization are considered, no significance can still be detected.

Miscellaneous. The total incidence of orchitis was 11.6 per cent, considering only those who developed this complication after entering the hospital. This is not significantly lower than that reported in the various larger series of cases. However, all our patients who developed orchitis had involvement of both parotid glands before they developed the orchitis. This might mean that they had to have a greater dissemination of virus before that complication developed. However, we only had 13 such cases, too few to make any generalization about this.

All our cases developed temperatures reaching 103° to 104° F. when the orchitis appeared. They appeared quite sick at this time. This is in harmony with other reports.[1, 3]

SUMMARY AND CONCLUSIONS

1. One hundred twenty-four cases of mumps occurring in soldiers are reported.
2. Belladonna was used in treating 42 of these cases without any effect on the duration of illness, incidence of complications, duration of orchitis, or comfort of the patient.
3. The distinction between rural and urban origin of the soldier is no longer as definite as it was in previously reported series of cases, probably due to decreased isolation of population groups.
4. Most of the men developed mumps within their first year of service and possible exposure and not in the first two months as reported in the World War I.
5. Early hospitalization does not shorten the duration of the disease.

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CASE REPORTS

SARCOIDOSIS WITH UVEOPAROTID FEVER *

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In 1889 Besnier [1] described peculiar lesions of the fingers, nose, and ears, a syndrome which he called lupus pernio. The sarcoid of Boeck,[2] described 10 years later, consisted of similar lesions of the skin, mucous membranes, and lymph nodes. In 1914 Schaumann [3] made an important contribution when he recognized that the lesions described by Besnier and by Boeck were actually manifestations of the same disease, which he called lymphogranuloma benignum in distinction to Hodgkin's disease, or lymphogranuloma malignum. During the next few years characteristic alterations of the lungs and of the bones were recognized and described by Kuznitsky and Bittorf,[4] and by Jüngling [5] respectively. The widely disseminated nature of sarcoid has been further emphasized in the extensive literature of the past 20 years; lesions have been described in almost every tissue of the body. It is now also recognized that, although unusual, Besnier-Boeck-Schaumann disease is by no means rare.

It is only in the past few years, however, that another obscure syndrome, the febris uveoparotidea subchronica described by Heerfordt [6] in 1909, has been generally regarded as a form of sarcoid. This entity is characterized by fever, bilateral uveitis and parotitis, and palsy of certain of the cranial nerves, most often the seventh. Bruins Slot [7] first suggested in 1936 that, because of the similar pathological picture and the occasional co-existence of Boeck's and Heerfordt's syndromes, uveoparotid fever is merely another form of sarcoidosis. Others, including Longcope and Pierson in this country,[8] have come to the same conclusion, and a number of cases of sarcoidosis with uveoparotid fever have been reported, chiefly in the ophthalmological literature.[9]

The purpose of this report is to describe a case in which the classical clinical features, both of sarcoidosis and uveoparotid fever, are combined in the same patient. This furnishes additional evidence of the identity of the two syndromes.

Case Report

Mr. G. H., a 25 year old negro shipyard worker, entered the medical ward of the Stanford University Hospitals on January 12, 1943, with the complaint of poor vision of two weeks' duration. Family and past history was not contributory. He had no dyspnea, fever, cough, fatigue or sputum and had no knowledge of exposure to tuberculosis or venereal disease. Five weeks before entry, while working without suitable welding goggles, he developed a steady, severe ache over both eyes. The company first aid worker said that he had received a flash burn, and gave him some

* Received for publication March 22, 1943.
From the Department of Medicine, Stanford University School of Medicine, San Francisco, California.

eye drops. Two weeks before entry he again noted pain in the eyes while at work and tried the same treatment. The next morning, however, the pain was unabated, and the patient noticed that the left side of his face was paralyzed so that he could not chew on the left, or close his left eye. Eye drops instilled by a private physician relieved his pain but the facial paralysis persisted, diminishing gradually during the next month. His vision became gradually worse, and he was finally seen in the out-patient eye clinic where he was found to have severe bilateral uveitis with synechiae and vitreous opacities, and admission to the hospital was arranged.

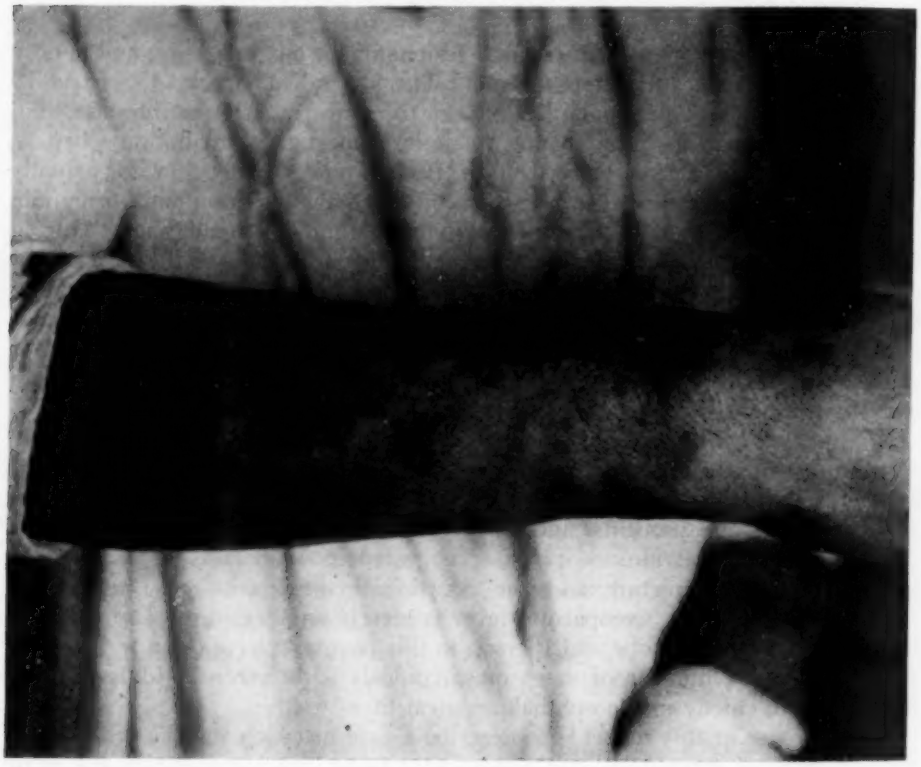

FIG. 1. Sarcoid infiltrations of the skin of the dorsum of the forearm.

Physical Examination. On admission the patient's temperature was 38.2° C., pulse 90, respirations 18, and blood pressure 116 mm. Hg systolic and 62 mm. diastolic. The patient was a well developed and nourished young negro who proclaimed himself completely well except for his eyes. The skin in general was moderately ichthyotic, but on the extensor surfaces of both forearms there were numerous 2–3 cm., soft, infiltrated, intracutaneous plaques, none of which had become scarred or depressed (figure 1). There was an infranuclear left facial palsy (figure 2). The pupils were very irregular, fixed to light, and dilated (patient was receiving atropine). Vision was 15/40 in either eye. Heavy, dense posterior corneal opacities could be seen by slit lamp, and there were numerous posterior synechiae. No areas of choroiditis could be seen in the fundus, and there was no increase in intraocular tension.

A firm, non-tender lymph node was palpable in the left pre-auricular region and there were shotty cervical nodes bilaterally; no generalized lymphadenopathy was made out. The breath sounds were somewhat louder over the left chest, but otherwise the heart, lungs, abdomen, and genitalia were not remarkable. The rectum contained no masses or strictures and the extremities and neurological findings were normal except as described.

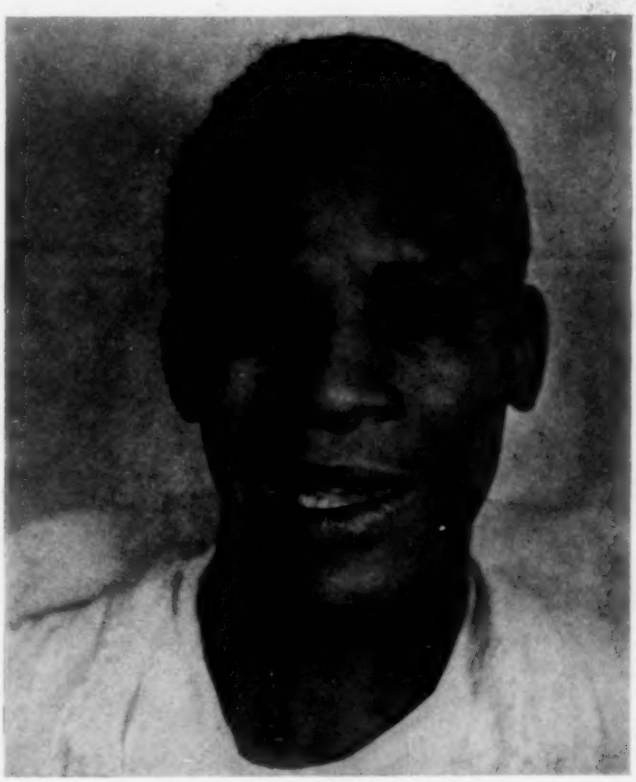

FIG. 2. Residual left facial palsy, one month after it first appeared. At the time of the photograph he was able to close his eye, but there was still definite weakness of the lower part of the face.

Laboratory Work. Blood count on entry showed 4.8 million red blood cells with 80 per cent hemoglobin and 5,600 white blood cells with 70 per cent polymorphonuclears of which 12 per cent were band forms, 20 per cent lymphocytes and 2 per cent monocytes. Total and differential white cell counts were essentially unchanged on three later occasions. Corrected sedimentation rate (Wintrobe) was 20, with a packed cell volume of 40. The urine was normal, and the Hinton and Wassermann reactions were negative.

Tuberculin tests with 0.01 mg. and 0.10 mg. of old tuberculin were negative, but a positive reaction was obtained with 1.0 mg. within 24 hours.

Roentgenogram of the chest (figure 3) showed enlarged hilar and mediastinal nodes with a diffuse granular infiltration of both lungs and these findings were unchanged in a second film taken two weeks later. Roentgenograms of the bones of

the hands, skull, tibiae, and feet showed no abnormalities. Partial gastrointestinal series was normal. Audiograms revealed no hearing defects.

Gastroscopy revealed no mucosal irregularities anywhere which might be interpreted as sarcoid infiltrations. Histological examination of the biopsy of one of the

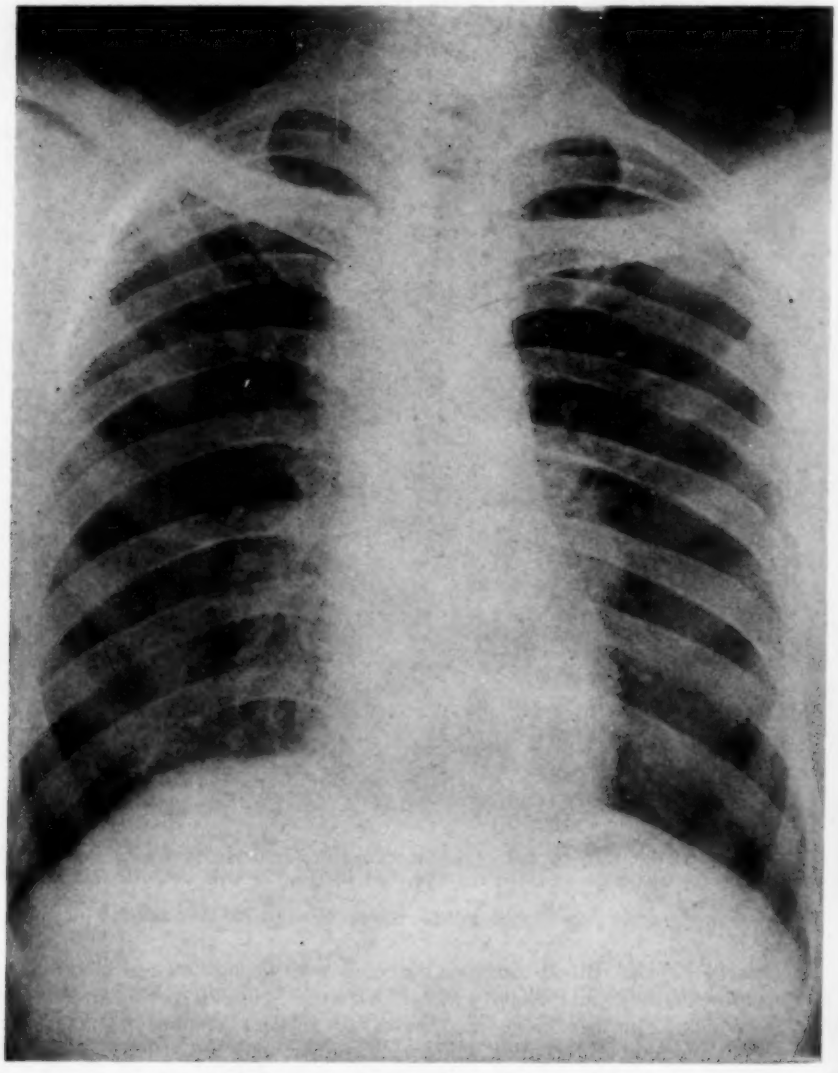

FIG. 3. Chest plate, showing enlargement of the hilar and mediastinal nodes, and granular infiltration of the lung fields.

skin nodules was reported as follows: "The sections of the skin show a stratified squamous epithelium with marked pigment in the basal layer of cells and a few stellate melanoblasts in the superficial corium. There is localized lymphocytic infiltration about the blood vessels and glands of the corium. Deeper in the corium are nodules of epithelioid cells about coiled glands. These can be seen grossly up to 1 mm.

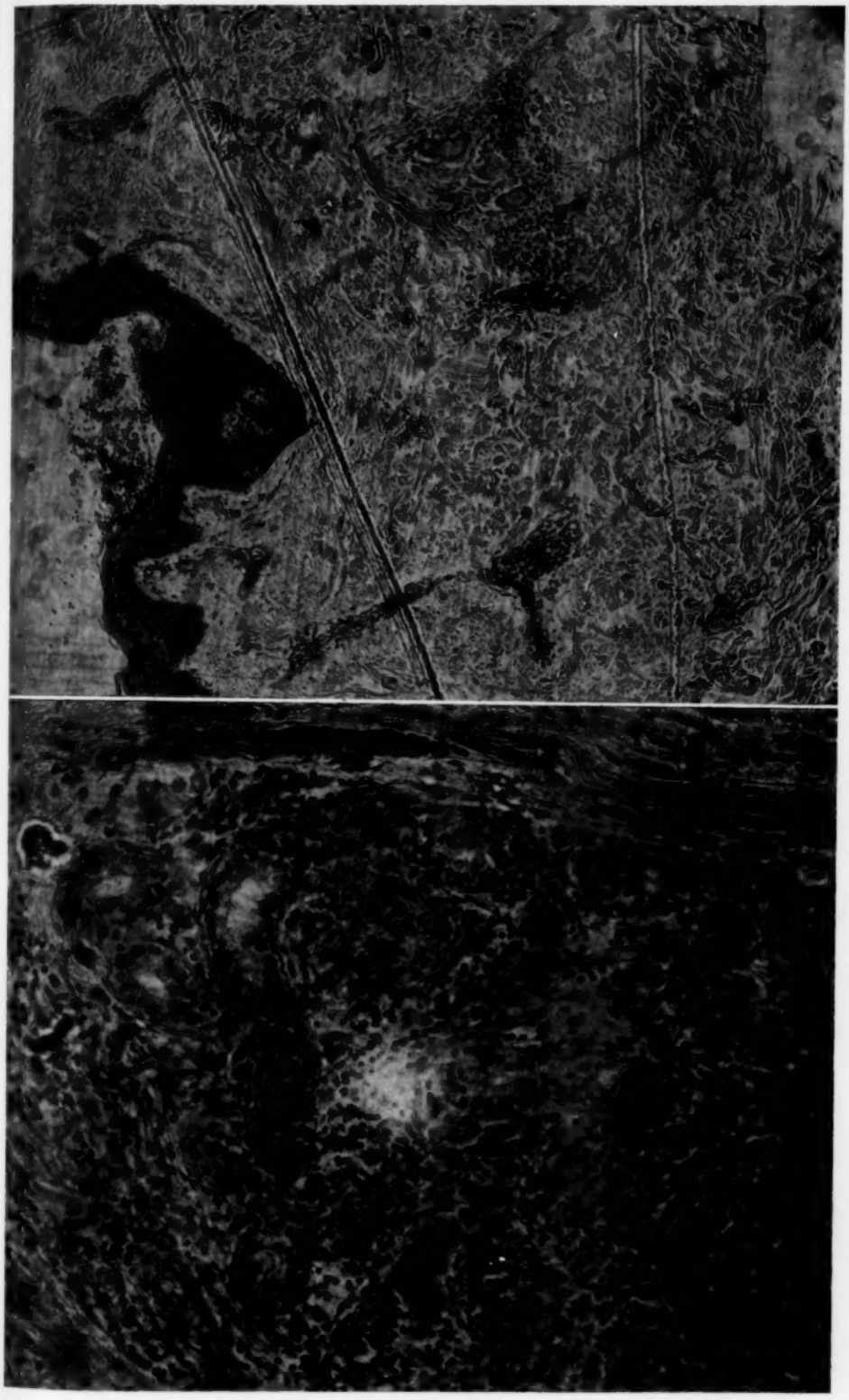

FIG. 4. Photomicrographs of the skin biopsy showing typical sarcoid infiltrations about the coiled glands, with giant and epithelioid cells surrounded by a lymphocytic reaction.

in diameter. Every nodule contains a coiled gland, but not every gland in the area has the proliferative reaction. Among the cells are lymphocytes and a few giant cells, some of which contain clear cytoplasmic vacuoles. The lining cells of the coiled glands appear normal in general, but between some of them are small lymphoid cells and in the cytoplasm of the epithelioid cells are irregular tiny acid fast globules singly or in clumps of 5 or 6." Photomicrographs of the sections are presented in figure 4.

On January 19, 1943, the blood cholesterol was 175.00 mg. per cent. The blood calcium was 10.3 mg. per cent. The blood phosphorus was 3.8 mg. per cent, and the blood phosphatase activity was 16.2 units, and 14 units on a later occasion. We are indebted to Dr. Eloise Jameson of Stanford University for performing electrophoretic studies of the serum proteins. The results on February 10, 1943, were total serum proteins 6.5 gm. per cent, albumin 3.38 gm. per cent, globulin 3.12 gm. per cent, with an A/G ratio of 1.09. The serum globulin percentages were as follows: alpha 0.56 per cent, beta total 22.16 per cent, gamma 25 per cent, and fibrinogen 0.28 per cent.

Course. During his stay in the hospital the patient's temperature ranged between 37.2 and 38.6° C. He had no specific complaints except for poor vision and pain in his eyes. Since his dismissal on January 19, 1943, he has been followed in the eye clinic where his vision has decreased progressively to 20/100 bilaterally. By February 11, 1943, the skin lesions had become flattened and in some cases scarred and the facial paralysis had disappeared. Except for poor eyesight, he has remained well and strong during a follow-up period of two months.

Comment

The classical features of both sarcoidosis and uveoparotid fever presented by this patient were skin nodules, adenopathy, pulmonary lesions, fever, parotitis, facial palsy, and bilateral uveitis. Failing vision and pain in the eyes were his only complaints; he did not notice the skin nodules on his forearms until they were called to his attention. It cannot be stated, therefore, whether the lesions of the skin and lungs had been present for some time, or whether they developed as part of a generalized process at the time he first noticed failing vision and facial paralysis. This young negro came recently from Tennessee to California to work in the ship yards, so that no inferences can be drawn regarding the geographical distribution of sarcoid. Although to our knowledge none has been reported, we are aware of at least one case of sarcoid and one of uveoparotid fever occurring in native Californians.

The laboratory studies were of interest. The blood count, performed during the febrile stage of the disease, was essentially normal, with no evidence of the eosinophilia or increase of mononuclear cells described in other cases. The sedimentation rate was at the upper limit of normal. The results of the electrophoretic studies of the serum proteins done four weeks after the patient's temperature had returned to normal showed the marked increase in beta, and especially in gamma, globulin reported by Fisher and Davis [10] as characteristic of the active stage of the disease. The serum calcium and phosphorus were normal but the phosphatase activity (alkaline) was surprisingly high in view of the absence of bone lesions found by roentgenographic examination.

SUMMARY

A striking case is presented in which the findings of both sarcoidosis and uveoparotid fever are combined. The classical features included skin nodules, adenopathy, pulmonary lesions, fever, parotitis, facial palsy, and bilateral uveitis. This patient offers convincing evidence of the identity of the two syndromes.

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RUPTURE OF ABDOMINAL AORTA INTO DUODENUM (THROUGH A SINUS TRACT CREATED BY A TUBERCULOUS MESENTERIC LYMPHADENITIS) *

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MESENTERIC adenitis presents a difficult diagnostic problem because of its diverse symptomatology. In abdominal laparotomies mesenteric adenitis of the non-tuberculous type is found in 6 per cent of cases [1] whereas the tuberculous

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variety is discovered in 0.74 per cent. General autopsy statistics reveal tuberculous mesenteric adenitis in 1–3 per cent; in patients with tuberculosis, the condition was discovered in only 0.79 per cent.

Anatomically the abdominal lymph nodes lie along the course of the arteries especially at those points where branches arise from the abdominal aorta. The tuberculous lymph nodes may appear merely enlarged, inflamed, and discrete. They may be found in groups or masses about a central caseous node. Finally only scarred peritoneum and calcified nodes attest to a latent stage of a previous infection. Most frequently those lymph nodes are involved which drain the ileocecal region, although any nodes are susceptible to this pathologic process.

From a study of the literature Colt and Clark [2] found that tuberculous lymphadenitis is often a fatal disease in the very young, but in those that survive and in older persons, the disease goes on to calcification and cure. Mead [1] reported that 55 per cent of his series go on to a five-year cure.

It is the caseous stage of tuberculous mesenteric adenitis which is most dangerous. Rupture of a caseous node is probably fairly frequent and causes tuberculous peritonitis. Whitmore [3] describes a case in which a large branch of the superior mesenteric artery had been eroded by the ulcerative process of tuberculous mesenteric glands. The bowel itself showed no sign of tuberculous ulceration; the only observable lesion was the adhesive peritonitis. Fischmann,[4] Ruescher,[5] and Rawitzkaja [6] have each described similar cases. In Rawitzkaja's report a 14 year old girl with tuberculous spondylitis developed a spurious aneurysm of a branch of the superior mesenteric artery due to erosion caused by a tuberculous mesenteric adenitis with rupture into the subserosa of the jejunum.

The present report concerns itself with an unusual complication of tuberculous mesenteric lymphadenitis and one the exact counterpart of which we have been unable to find in the literature.

Case Report

F. G., a 51 year old white male, station agent by occupation, was well until the age of 39 years when he was admitted to Sea View Hospital in 1928 for an osteomyelitis of the left humerus and shoulder. He remained in the hospital for 14 months, but the infection was never proved to be due to the tubercle bacillus.

Three years later, in 1931, he was a patient at Morrisania City Hospital for one month for lobar pneumonia. An electrocardiogram at this time showed a sinus tachycardia with myocardial disease.

In 1934 he was readmitted to Morrisania City Hospital with complaints of urinary retention, enlarged left testicle and a discharging sinus above the scrotum. The clinical diagnosis of tuberculous epididymitis was confirmed by microscopic examination after epididymectomy and vasectomy were done. He was discharged from the hospital as improved two months later.

During the next six years the patient was apparently well until July, 1940, when he complained of sudden epigastric pain and vomited bright red blood. Immediately following admission he vomited a pint of bright red blood. There were no other complaints and no previous history of gastric disturbance.

Examination revealed a pale, white male, appearing considerably older than 51 years, acutely ill, not restless, with no air hunger. Pulse was 100 per minute; blood pressure was 94 mm. of mercury systolic and 58 mm. diastolic. Temperature was 99° F. The heart was somewhat enlarged to the left; the apex beat was neither seen nor

felt and the heart sounds were distant and regular. The abdomen was soft, moderately distended, without tenderness. A suprapubic cystostomy scar was visible. The liver and spleen were not palpable. On the left shoulder was the scar of an osteomyelitis. A dressing over the sternum covered several draining sinuses. There was no clubbing of the fingers.

FIG. 1. Perforation in aorta.

The red blood cell count ranged from 3,000,000 to 3,200,000 and the hemoglobin from 64 to 70 per cent. Urine contained 2 plus albumin with 10 to 12 white blood cells per high power field. Blood serologic test for syphilis was negative. The Congo red test gave 38 per cent absorption. Stool was strongly positive for blood.

A clinical diagnosis of a bleeding peptic ulcer was made because of the presence of the bright red blood in the vomitus. The possibility of ruptured esophageal varices was entertained; this being secondary to cirrhosis of the liver on an amyloidosis basis as a result of the longstanding chronic infection. This was ruled out by the normal findings in the Congo red test.

However, fluoroscopic and radiographic examination of the esophagus, stomach, and duodenum showed no evidence of an organic lesion. Examination of the chest revealed a thickening of the pleura and retraction of both apices. Roentgenographic studies of the right sterno-clavicular joint and left shoulder revealed an infectious process involving the sternal end of the first rib, the inferior border of the sternal end of the right clavicle, and partial destruction of the head of the humerus and glenoid fossa.

On a Sippy régime his condition improved and he was discharged as improved one month after admission with a diagnosis of bleeding peptic ulcer. While on the ward he had had no further hematemesis.

The day following his discharge from the hospital he vomited about 500 c.c. of bright red blood and was readmitted. He appeared acutely ill, pale and cyanotic. Pulse was 100 per minute; blood pressure was 95 mm. of mercury systolic and 60 diastolic. Temperature was 97.6° F. The abdomen was soft, but tenderness was elicited in both lumbar regions. After a transfusion of 500 c.c. of citrated blood and the usual supportive measures he remained comfortable for one week. On September 5, 1940, he complained of severe stabbing epigastric pain and vomited bright red blood. He became ashen, pulseless and ceased breathing one hour later.

FIG. 2. Perforation in duodenum.

Postmortem Examination. Postmortem examination by Dr. Jacob Taub revealed fibrotic lung tissue but no evidence of caseation or tuberculosis. The abdominal viscera were extremely pale. The stomach was dilated and filled with approximately 1000 c.c. of clotted blood. The pyloric opening and duodenum were also filled with blood. In the lower third of the duodenum a punched out perforation, one-fourth of an inch in diameter and containing thrombotic material, was discovered. A probe was passed through this perforation into a sinus, one-half inch in length, communicating directly with the abdominal aorta which in this region showed extensive, ulcerative, atheromatous plaques. The communicating sinus contained a well-formed, grayish, friable thrombus. Cross-section of the abdominal aorta and duodenum in this region showed them to be separated from each other by a tuberculous lymph node, the size of a marble, through which the communicating sinus passed.

The jejunum and ileum were also filled with clotted blood. The only other significant findings were in the kidneys. The right kidney was reduced to one half normal size and contained a pasty, cheese-like material which completely filled and dilated all the calyces. The left kidney was increased in size probably because of compensatory hyperplasia. The ureters and bladder showed no gross disease.

SUMMARY

A case of tuberculous, caseous, mesenteric lymphadenitis is presented. One large mass of caseous lymph glands had apparently eroded the abdominal aorta and formed a small opening, through which blood burrowed slowly forming a sinus tract which led to the third portion of the duodenum into which it ruptured, leading to several hemorrhages. The first two were apparently stopped by a small plug closing the opening temporarily; the last one was exsanguinating.

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LARGE INTERAURICULAR SEPTAL DEFECT ASSOCIATED WITH TUBERCULOSIS AND AMYLOIDOSIS*

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A DEVELOPMENTAL defect of the interauricular septum may vary from a small patency of the foramen ovale to complete arrest of formation of the septum. When the interauricular communication is one or more centimeters in diameter the defect may be considered sufficiently large to permit a considerable shunt of blood from the left to the right side of the heart. As a result, there ensues a significant alteration in circulatory dynamics with consequent anatomic changes in the heart and pulmonary vessels. Modern contributions to the knowledge of interauricular septal defects have been made by Abbott,[1] Assman,[2] Roesler,[3] McGinn and White,[4] Tinney[5] and others so that the embryological, pathological, roentgenographic and clinical features of this lesion are now well correlated.

Although small anatomical defects of the septum are of frequent occurrence, large interauricular defects are relatively rare. This has been indicated by

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Roesler [3] who, in a most comprehensive review of the literature from 1826 to 1933, was able to collect only 62 cases in which the communication between the auricles was widely patent and not complicated by other cardiac anomalies. The following important features of large defects of the interauricular septum have been noted: (1) The hearts are always large; often they are enormous. This enlargement, even in the absence of a valvular lesion, is due entirely to dilatation and hypertrophy of the right side and is often influenced by the size of the interauricular defect. (2) The aorta is small or may be normal, whereas the pulmonary artery is always larger and together with its branches shows pronounced arteriosclerosis. (3) The densities of the enlarged pulmonary arteries and their branches have led often to an erroneous roentgen diagnosis of pulmonary tuberculosis, which in actuality occurs rarely. (4) Auricular fibrillation is common in contradistinction to other cardiovascular malformations in which auricular fibrillation is rare. (5) The clinical course leading to death is usually that of cardiac failure and the average duration of life is 36 years.

The case reported here presented a rather large defect of the interauricular septum complicated by the presence of chronic advanced pulmonary tuberculosis. The clinical course was featured by recurrent attacks of paroxysmal auricular tachycardia with cardiac decompensation, the development of advanced generalized amyloidosis, and the terminal occurrence of hypertension, uremia and pulmonary embolism.

Case Report

C. L., a white female clerk, 21 years of age, was first admitted to the Hudson County Tuberculosis Hospital on August 29, 1932. Her family history was negative. There was nothing significant in her childhood history, and there was no knowledge of the presence of heart disease. At the age of 19 she developed a cough, several small hemoptyses, and recurrent pleurisy. She did not seek medical attention until two years later when the symptoms of fatigue, weight loss, dyspnea and cardiac palpitation on exertion appeared. A diagnosis of pulmonary tuberculosis was established and admission to the hospital followed. At this time she appeared chronically ill and moderately undernourished; her weight was 86 pounds. Examination of the thorax revealed signs of a bilateral pulmonary infiltration with excavation of the right upper lobe. There was enlargement of the heart, and a marked apical systolic thrust was visible. A coarse systolic thrill was palpable over the pulmonic area and a rough loud systolic and diastolic murmur was heard over this zone. At the apex there was a soft, blowing systolic murmur. The aortic sounds were not distinguishable. The cardiac rhythm was regular with an apical rate of 110 per minute. The peripheral pulses were small. The systolic blood pressure was 102 mm. of mercury and the diastolic 78. Cyanosis and clubbing of the fingers were not present.

A roentgenogram (figure 1) of the chest disclosed a bilateral bronchopneumonic type of infiltrate affecting principally both upper lung fields with a large area of excavation in the right upper lobe. The cardiac silhouette was large and globular with a pronounced bulge of the pulmonary conus and a small aortic arch. There was pronounced accentuation of the bronchovascular hilar markings.

Aside from the presence of tubercle bacilli in the sputum all other laboratory findings were within normal limits at this time.

The diagnosis on admission was congenital heart disease and bilateral, chronic pulmonary tuberculosis with cavitation of the right upper lobe. The nature of the cardiac abnormality was considered to be either an interauricular or interventricular septal defect, or a patent ductus arteriosus.

An attempted therapeutic pneumothorax on the right was unsuccessful because of adherent pleurae and a right temporary phrenic nerve interruption was performed in October 1932 without any effect upon the cavity. With restriction of physical activity there was roentgenographic improvement of the pulmonary lesion, and the clinical course remained uneventful for a period of two years. On July 23, 1934 she was seized suddenly with a rigor, elevation of temperature to 104° F., chills, orthopnea and prostration. On examination there was marked skin pallor and cyanosis of the

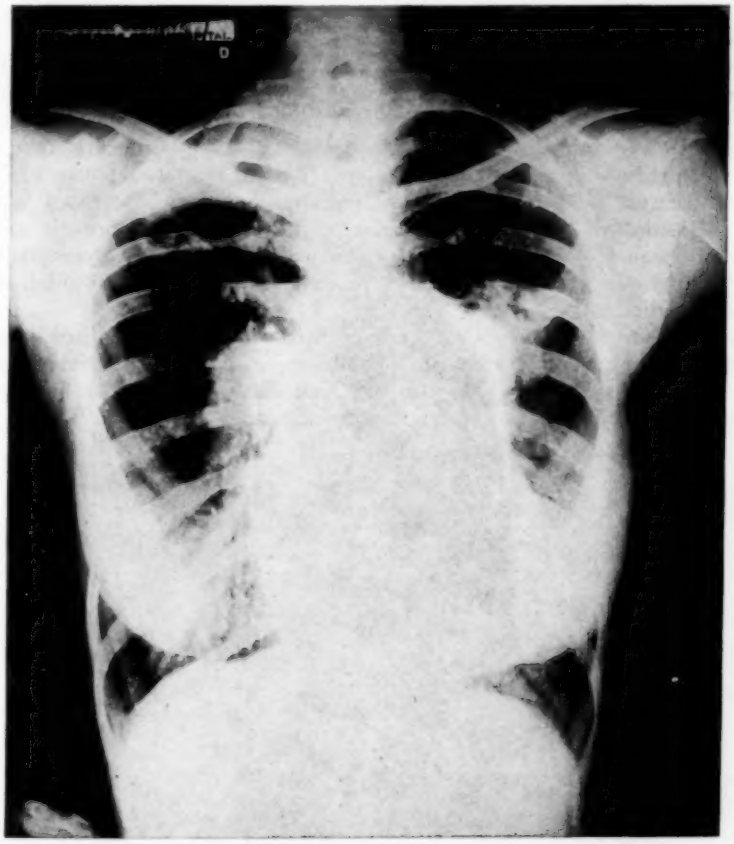

FIG. 1. Roentgenogram of chest showing the large globular heart, the small aorta, the pronounced accentuation of the pulmonary conus, the densities of the enlarged pulmonary arteries, the pulmonary changes due to the lesions of tuberculosis.

lips and nailbeds. Moist râles were heard throughout both lung fields. The heart murmurs were accentuated and the heart action was pounding; the rate was 160 per minute and the blood pressure was unchanged. Roentgenographic examination of the chest at this time revealed a marked dilatation of the heart and diffusely broadened bronchovascular hilar markings. Rapid digitalization was instituted and within 4 days the patient showed significant improvement. However, from this time on the patient was dyspneic on exertion. On a few occasions she was permitted to follow a home regimen but in the main was restricted to a semi-invalid existence in the hospital. The tuberculous involvement of the lung showed occasional mutation of the infiltration with calcification in scattered areas. There was a gradual increase in the

size of the heart as well as the pulmonary vessels and on fluoroscopy the pulsations of the latter were easily discernible well out into the fields of the parenchyma. Electrocardiograms showed extreme right axis deviation, and heart sound tracings confirmed the character of the murmurs. The patient suffered recurrent attacks of paroxysmal auricular tachycardia which were controlled with quinidine.

Albuminuria appeared during this period (1934) and thereafter persisted. The presence of amyloidosis was confirmed by the Benhold congo red test in 1936 at which time hepatomegaly and occasional slight dependent edema were demonstrable. Chemical examination of the blood revealed hypoproteinemia with reversal of the albumin-globulin ratio.

For the next five years there was persistent edema of the lower extremities with a small amount of ascites and transient facial edema. Finally in 1941 hypertension appeared for the first time; the systolic blood pressure was 154 mm. of mercury and the diastolic 100. Examination of the blood also showed for the first time elevation of the urea nitrogen. These findings were ascribed to the development of secondary contraction of the amyloid kidneys. Thereafter anasarca increased slowly. In October 1941 the patient was conscious of increasing intrathoracic discomfort. Orthopnea became more marked and the skin pallor and digital cyanosis increased. On October 6, 1941, nine years after her first admission into the hospital, she lapsed into coma and died.

Autopsy Findings. At postmortem examination there was marked cyanosis and some edema of the lower extremities. The abdominal cavity contained about 500 c.c. of clear, amber colored fluid. There were about 200 c.c. of similar fluid in the left pleural cavity. The pleurae on the right were adherent.

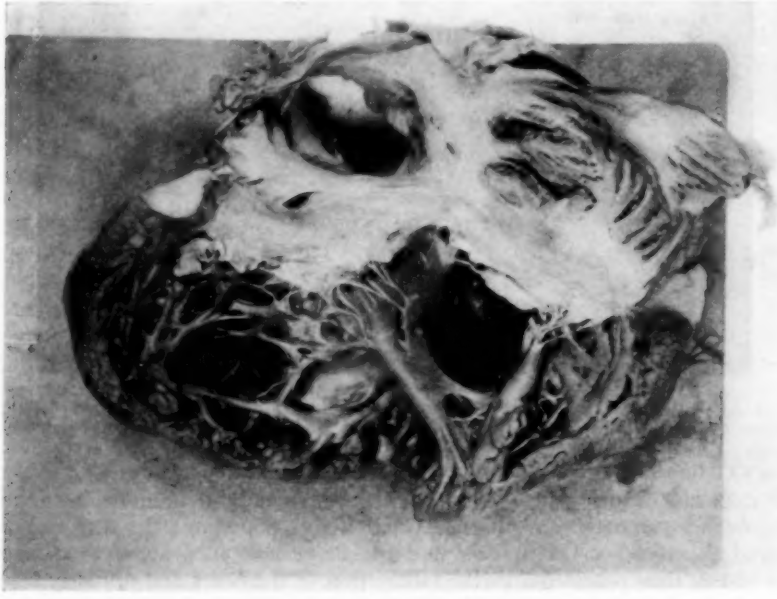

FIG. 2. Right ventricle opened to show enormous auricular and ventricular hypertrophy and dilatation. Note large interauricular septal defect.

Heart (figure 2). There was no excess of fluid in the pericardial cavity. The heart weighed 440 gm. There was a marked enlargement of the right ventricle which made up the entire anterior aspect of the heart. This enlargement was due to both

hypertrophy and dilatation. The left ventricle was of normal thickness and slightly dilated. The myocardium of both ventricles was firm and grayish red. There was marked enlargement of the right auricle, with pronounced hypertrophy and dilatation of the right auricular appendage. The posterior wall of the auricle was occupied by an oval orifice measuring 5 cm. in diameter. At the inferior margin of this defect there was a small ridge of tissue lying just above the site of insertion of the tricuspid valve. Laterally and superiorly the right and left auricular walls blended except for a second small fold about 3 mm. high at the site of entrance of the right pulmonary

FIG. 3. Posterior view of right lung showing enormous dilatation of pulmonary artery branches and emboli to upper lobe.

vein into the left auricle. The endocardium of the right auricle was somewhat more opaque than that of the left, thereby aiding in the demarcation of the two auricles. The leaflets of the tricuspid valve were moderately thickened, particularly along the closing margins. The trabeculae carneae of the right ventricle were thick and broad. The ostium of the pulmonary artery was enormously dilated and measured 8 cm. in circumference when opened. The pulmonary valves appeared competent. The leaflets were somewhat thickened and incipient fusion of the commissures was noted. There was enormous enlargement of the pulmonary conus. The left auricle

was small and consisted of segments of anterior and posterior wall together with the left auricular appendage. On the posterior wall there was a small thrombotic mass 2 by 3 mm. The mesial wall was formed by the defect described above; its base rested upon the superior margin of the mitral ring. The leaflets of the mitral valve were thickened and fused. The chordae tendineae were somewhat thickened and shortened. The aortic valve was competent and the cusps were translucent. The aorta was of normal size and measured 4 cm. across when opened. The coronary ostia and the lumina of the arteries were patent; the intima of the coronary arteries was smooth. The venae cavae were slightly dilated at the point of entrance into the right heart, but otherwise appeared normal.

Lungs (figure 3). Occupying the mid-portion of the left upper lobe there was a circular thick walled tuberculous cavity about 5 cm. in diameter. A similar cavity approximately 7 cm. in diameter was present in the right upper lobe. Scattered throughout both lungs, particularly in the upper lobes, there were many well circumscribed old and some more recent tuberculous nodules 2 to 4 mm. in diameter. There was moderate emphysema and many small calcified nodules were palpated throughout both lower lobes and to a lesser extent in the upper lobes. The main pulmonary artery and its branches were dilated to several times their normal diameter. This dilatation extended to the finest ramifications and many of the vessels were markedly thickened along their entire course. A firm grayish, somewhat adherent embolus completely occluded the lumina of the main branches to both upper lobes. Embolization was seen in some of the smaller ramifications in the lower lobes as well. The intimal surface of the main artery and its branches showed many scattered, elevated, irregular, yellowish atheromatous plaques along the entire course. The distribution of the vascular thickening was somewhat irregular. On the whole the vessels in the lower portions of the upper lobe and throughout the lower lobes were more involved. The vessels to the upper portion of the upper lobes appeared to be spared.

The liver and spleen both presented gross evidence of amyloid disease.

The kidneys were of equal size, firm, yellowish red and each weighed 115 gm. Upon stripping the capsules the surfaces showed diffuse granularity with numerous small pitted areas. On section there was some narrowing of the cortex. The corticomedullary differentiation was not very distinct. Many of the interlobular vessels stood out prominently.

There was congo red staining of the liver, spleen and kidneys. (The last congo red test was performed seven months before death.)

The essential microscopic findings were as follows:

Heart. The left auricular wall showed proliferation of the subendothelial connective tissue with elastica reduplication. There were scattered partially organized thrombotic masses on the endocardial surface. These did not contain bacteria. The right ventricle showed focal areas of increased connective tissue with some atrophy of the muscle fibers. The increase in connective tissue was not limited to any specific area. There was hypertrophy of the muscle fibers throughout.

Pulmonary Artery. A section through the main pulmonary artery showed a mild degree of intimal proliferation. The media was not significantly altered. The elastica was intact. In the adventitia some of the vasa vasorum presented a mild periarterial infiltration of round cells.

Lungs. The sections through the tuberculous cavities in both upper lobes showed the characteristic appearance of old cavity with fibrosis and organization of much of the surrounding pulmonary parenchyma. The branches of the pulmonary artery presented varying degrees of dilatation, intimal proliferation and medial hypertrophy (figure 4). The larger vessels were rather markedly dilated and showed occasionally pronounced intimal proliferation with subintimal fat deposits and atrophy and scarring of the media. The smaller vessels disclosed a more pronounced degree

of intimal proliferation with elastica reduplication attended by varying degrees of narrowing of the lumina. One of the outstanding features was the rather marked variation in the distribution of the vascular alterations. The right upper lobe appeared to be the least involved and multiple sections revealed only occasional vessels with intimal proliferation and medial hypertrophy. The lower lobe showed a great degree of vascular involvement. The left lung on the other hand revealed considerable

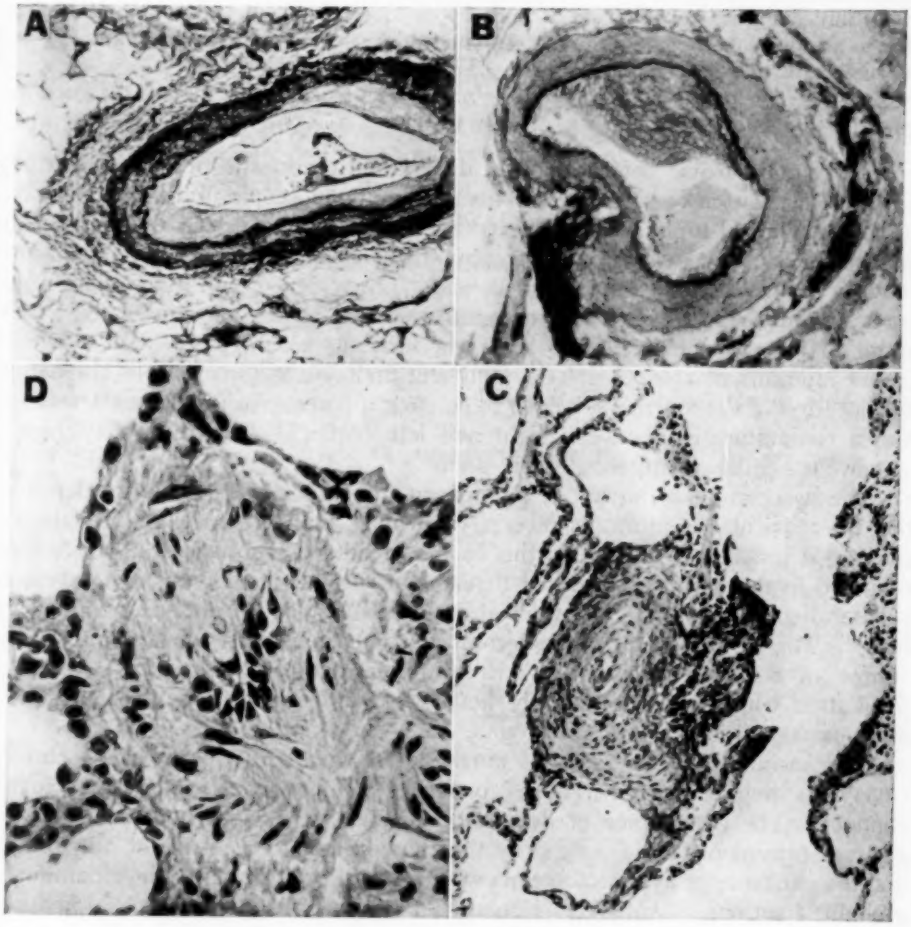

FIG. 4. *A and B,* medium sized artery branches. Elastica stain. Note intimal proliferation. *C,* low power view of arteriole showing striking medial hypertrophy with remarkable reduction in lumen. *D,* high power view of another arteriole showing similar features.

vascular changes in the upper lobe and a pronounced involvement of the smaller branches in the lower lobe. Nevertheless, even in this lung the distribution of the vascular lesions was not uniform. Very occasionally a small arteriole was seen to be filled by a partially organized thrombus.

Kidneys. The kidneys showed extensive amyloid infiltration involving all the glomeruli, with much scarring and glomerular and tubular atrophy. The vessels of the kidneys showed exceedingly little reaction aside from amyloid infiltration.

The liver, spleen and adrenals presented moderate amyloid infiltration. The remaining organs were without histological significance.

Anatomical Diagnosis. (1) Congenital heart disease: Large defect of the interauricular septum; enormous hypertrophy and dilatation of the right auricle and right ventricle. Marked dilatation of the pulmonary conus, main pulmonary artery and branches with moderate arteriosclerosis. Slight dilatation of the left ventricle. Interstitial mitral valvulitis. (2) Pulmonary embolism of both upper lobe branches. (3) Fibrocavernous tuberculosis of both upper lobes, with old and recent dissemination to all lobes. Emphysema. (4) Amyloid disease of the liver, spleen, kidneys and adrenals; amyloid contracted kidneys. (5) Anasarca.

COMMENT

Congenital cardiac defects provide differential diagnostic problems in which a careful evaluation of signs, symptoms and roentgenoscopic findings is essential to correct clinical impression. The roentgenoscopic evidence in this case of an enlarged globular heart with hypertrophy of the right chambers, a prominent pulmonary conus, a small aorta, and considerably widened pulsating hilar structures (pulmonary arteries) radiating into the lung parenchyma indicated a vast shunt of blood from the left to the right heart as would occur with a widely patent interauricular septal defect. Physical findings, as for example, the nature and location of the murmurs, helped to rule out a patency of the ductus arteriosus and a communication between right and left ventricles, conditions in which a somewhat similar roentgenogram is seen.

The association of advanced pulmonary tuberculosis with a large defect of the interauricular septum is rare. A chronic, intermittently active bilateral cavernous lesion was present in this case. Healing by fibrosis and calcification occurred in the infiltrate scattered throughout the lung fields, and the extensive collateral emphysema which developed undoubtedly added to the load of the right heart. The evidences of cardiac decompensation, however, were seen only in the course of the severe bouts of paroxysmal auricular tachycardia. Auricular fibrillation which is the commonly noted arrhythmia with widely patent interauricular septal defects did not occur.

The insidious development of amyloidosis which often complicates chronic cavernous pulmonary tuberculosis produced a number of important clinical changes. The appearance of dependent edema, ascites and hepatomegaly was early disproved of cardiac origin by the Benhold congo red test for amyloidosis and the existence of hypoproteinemia with reversal of the ratio of the albumin and globulin fractions. Moreover circulation time and venous pressure estimations yielded normal values. The renal contraction incident to the amyloid nephrosis further added to the burden of the heart in the appearance of hypertension. Terminal azotemia appeared as a complicating feature.

The presence of pulmonary emboli in instances of interauricular septal defect has been mentioned. It is also noteworthy that in many instances of amyloidosis pulmonary embolism is commonly encountered. Undoubtedly the latter was the factor which contributed to the occurrence of the fatal pulmonary embolism in this case.

The anatomical findings in the heart and lungs were comparable to those frequently described in instances of widely patent interauricular septal defects. One of the rather interesting and as yet unexplained features was the irregular

distribution of the pulmonary arteriosclerosis. This lesion appeared to be most extensive in the lower lobes and lower portion of the upper lobes. Even within the more involved segments of the lung the arteriosclerotic changes were not uniform. There was no distinctive relationship to the associated lesions of tuberculosis.

Conclusions

An instance of a very large interauricular septal defect of congenital origin is described. The case was complicated by the rare presence of bilateral upper lobe cavernous tuberculosis with amyloid nephrosis and incipient renal contraction. Terminal hypertension and azotemia contributed to the decline of the patient who finally died with pulmonary embolism.

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4. McGinn, S., and White, P. D.: Interauricular septal defect associated with mitral stenosis, Am. Heart Jr., 1933, ix, 1.
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EDITORIAL

CEREBRAL VASCULAR LESIONS IN RHEUMATIC FEVER

ALTHOUGH it has long been known that rheumatic fever is a generalized infection in which many tissues and organs may be involved, the frequent occurrence of disseminated vascular lesions in this disease has not been so generally appreciated. Krehl [1] is generally credited as the first clearly to describe such lesions in the myocardium of patients with rheumatic valvular disease. His observations have been amply confirmed and the character of the lesions described, particularly by Karsner and Bayless and in great detail by Gross, Kugel and Epstein.[2] The latter observed outspoken endarteritis in one-third of their active cases.

Such lesions, however, are by no means limited to the coronary vessels. The widespread distribution of vascular lesions was emphasized by Von Glahn and Pappenheimer [3] in 1926. They reported finding lesions in the peripheral vessels of 10 of 47 consecutive cases of rheumatic fever. Aside from the myocardium, lesions were found in the lung, aortic valve, kidney, perirenal and periadrenal fat, ovary, testis, pancreas, appendix epiploica of the sigmoid colon and a small polyp of the cecum. To this list must be added, among others, the meninges and cerebral cortex.

The detailed histological structure of these lesions varies considerably, depending in part upon the acuteness and duration of the process. In well marked cases there is an endarteritis which, according to Von Glahn, starts with swelling, proliferation and exfoliation of the intimal endothelium, but without thrombosis. There is swelling of the vessel wall with edema and infiltration with fibrin. Peripherally he described a loose fibrillar stroma containing many nuclei, both lobulated nuclei of polymorphonuclear leukocytes and oval vesicular nuclei which tended to assume a radial arrangement about the vessel. Necrosis of cells sometimes occurred. The internal elastic lamella became swollen, shredded and broken. Later there was sometimes partial canalization by ingrowth of vascular endothelium, or the fibrin was replaced by fibrillar connective tissue, resulting in an obliterating endarteritis. The capillaries were also involved.

These changes result in local obstruction to the circulation. The tissues supplied by the diseased vessels suffer in varying degree. There may be cellular atrophy, or focal necroses, with cystic areas of softening.

Although Von Glahn [3] and Gross [2] thought the lesions were in some degree specific for rheumatic fever, subsequent investigators agree that

[1] KREHL, L.: Beitrag zur Pathologie der Herzklappenfehler, Deutsch. Arch. f. klin. Med., 1890, xlvi, 454.

[2] GROSS, L., KUGEL, M. A., and EPSTEIN, E. Z.: Lesions of the coronary arteries and their branches in rheumatic fever, Am. Jr. Path., 1935, xi, 253.

[3] VON GLAHN, W. C., and PAPPENHEIMER, A. M.: Specific lesions of peripheral blood vessels in rheumatism, Am. Jr. Path., 1926, ii, 235.

anatomically they can not be distinguished from endarteritis caused by other infections, and particularly not from syphilitic endarteritis.

Perhaps the first to describe such lesions in the brain were Winkelman and Eckel,[4] who in 1929 reported the case of a woman who developed a severe psychosis a few months after an attack of acute rheumatic fever. At autopsy shortly afterward they demonstrated a proliferative endarteritis of the small cortical vessels and minute areas of softening in the gray matter. In 1932 they described brain lesions, some of them similar in character, in five other cases.

This subject has since been studied intensively by Bruetsch,[5] who has recently published a summary of his observations. His attention was attracted to the subject by the fact that in routine autopsies carried out in a large psychiatric hospital, patients with rheumatic valvular disease frequently showed lesions in the brain which were regarded as rheumatic in origin. In 500 consecutive autopsies rheumatic valvular disease was present in 5 per cent of the cases. In 100 cases of schizophrenia the incidence was 9 per cent. Most of these patients had been inmates for many years, and none was known to have had rheumatic fever during his stay in the institution.

A study of about 500 cases each of male and female psychiatric patients, representing consecutive admissions to the hospital, showed that 2.6 per cent of the males and 8.1 per cent of the females either showed clinical evidence of rheumatic valvular disease or gave a history of acute rheumatic fever. It is manifestly possible that an individual with chronic rheumatic endocarditis may develop a psychosis as a result of purely psychogenic disturbances. However, in view of the fact that the incidence of rheumatic infection in the general population is generally estimated to be somewhat less than 1 per cent, these figures appear to be statistically significant, and suggest a direct relationship between the brain lesions and the psychosis in many of the cases.

Bruetsch recognizes three types of brain involvement: an obliterative endarteritis, a meningoencephalitis (rare), and cerebral embolism. The occasional occurrence of cerebral embolism in cases of mitral stenosis with auricular fibrillation, as well as in those with a complicating bacterial endocarditis has long been recognized. The endarteritic cerebral lesions are of more immediate interest. In Bruetsch's cases brain lesions were very frequent. Of the 30 cases showing rheumatic valvular disease, brain lesions were found in all but one. In 15 cases there were gross infarctions, and in 14 there were microscopic areas of softening in the cortex. Lesions in other parts of the brain were rare.

The character of the lesions in the cerebral arteries did not differ materially from those described in other organs. In most cases the patients died many years after the rheumatic infection had been established, and the

[4] WINKELMAN, N. W., and ECKEL, J. Z.: Endarteritis of the small cortical vessels in severe infections and toxemias, Arch. Neurol. and Psychiat., 1929, xxi, 863.

[5] BRUETSCH, W. L.: Late cerebral sequelae of rheumatic fever, Arch. Int. Med., 1944, lxxiii, 472.

cortical vessels showed largely the end stages of an obliterative endarteritis. In a few cases, however, autopsy was obtained shortly after the onset of the psychosis, and revealed early endarteritic changes with cystic areas of softening in the cortex. Even in the long standing cases, however, decades after the onset of the illness, vessels were sometimes found in which there were evidences of acute inflammation, indicating the persistence of active infection throughout this period.

The type of psychosis presented by these patients was not specific of rheumatic fever, but apparently depended upon the personality of the individual and the age at onset. In many of the younger adults it was a schizophrenia. In some of the older cases it was an involutional depression. In children it might appear as feeblemindedness or as behavior disorders. The possible relationship of vascular changes of this type to chorea is an interesting problem which is not yet settled. Endarteritis of the cerebral vessels in chorea has been reported by von Sántha.[6] The fact that in chorea clinical recovery is usually complete, however, even if choreiform disturbances have been severe, suggests that the lesions are quantitatively less severe, if not qualitatively different.

Rheumatic brain disease may manifest itself clinically in other ways. Epileptiform convulsions may occur.[7] Foster [8] has pointed out that seizures of this type are several times as common in individuals with rheumatic heart disease as in the general population. One case has been reported by Alexander [9] with the clinical manifestations of epidemic encephalitis in which (exceptionally) the vessels of the basal ganglia were involved. A hemiplegia resulting from endarteritis of a larger vessel would be another possible manifestation.

Bruetsch's observations emphasize the fact that rheumatic fever is a protracted chronic infection, the duration of which must often be measured by years and even decades. The cerebral lesions apparently do not materially shorten life. The possibility of any significant improvement after symptoms of cerebral involvement have appeared, however, seems small, either with or without specific treatment. The most that can be hoped for is possibly to reduce the incidence and severity of such lesions by early and protracted treatment of the infection. More detailed clinical studies of these cases are needed, particularly with regard to fever, sedimentation rate and electrocardiographic changes.

[6] VON SÁNTHA, K.: Über Gefässveränderungen im Zentralnervensystem bei Chorea rheumatica, Virchow's Arch. f. path. Anat., 1932, cclxxxvii, 405.

[7] BREUTSCH, W. L.: Rheumatic epilepsy: sequel of rheumatic fever, Am. Jr. Psychiat., 1942, xcviii, 727.

[8] FOSTER, D. B.: Association between convulsive seizures and rheumatic heart disease, Arch. Neurol. and Psychiat., 1942, xlvii, 254.

[9] ALEXANDER, L.: The diseases of the basal ganglia, Proc. Assoc. Research Nerv. and Ment. Dis., 1942, xxi, 454.

REVIEW

Starling's Principles of Human Physiology. Edited and revised by C. LOVETT EVANS, D.Sc., F.R.C.P., F.R.S., LL.D. Birmingham, Jodrell Professor of Physiology in University College, London. Chapters of the special senses revised by H. Hartridge, M.A., M.D., Sc.D., F.R.S., Professor of Physiology at St. Bartholomew's Medical College. 8th edition. 1,247 pages; 24.5 × 16 cm. Lea and Febiger, Philadelphia. 1941. Price, $10.00.

Dr. Evans has revised, rearranged, and practically rewritten the previous edition without, however, changing to any marked degree the readable style which has been a characteristic of this textbook. The point of view of the editor remains scientific rather than clinical, although clinical material of general interest has been used freely. The greatest changes have been made in the chapters pertaining to the central nervous system and the special senses. These have been completely revised to bring them in line with the newer work in these fields. Because of the extensive changes in the fields of endocrinology and reproduction, these sections have also been altered to a great extent. Although it is recognized that it is difficult to select material for a text of this kind, it would appear that somewhat more emphasis could have been put on the chapters on nutrition, particularly on vitamins and normal diet. The volume is exceptionally well illustrated. A number of the figures were drawn especially for this edition; others have been copied from the literature.

The index is very complete. Various sections of the text are followed by partial bibliographies, and specific references in the text are given in footnotes.

The editor and the publishers are to be commended for the careful preparation of this volume.

M. A. A.

BOOKS RECEIVED

Books received during July are acknowledged in the following section. As far as practicable, those of special interest will be selected for review later, but it is not possible to discuss all of them.

Heart Disease. Third Edition. By PAUL DUDLEY WHITE, M.D. 1,025 pages; 22 × 14.5 cm. 1944. The Macmillan Company, New York City. Price, $9.00.

Technic of Electrotherapy and Its Physical and Physiological Basis. By STAFFORD L. OSBORNE, M.S., Ph.D., and HAROLD J. HOLMQUEST, B.S., B.S.(M.E.) 780 pages; 23.5 × 15 cm. 1944. Charles C. Thomas, Springfield, Illinois. Price, $7.50.

Experimental Basis for Neurotic Behavior. By W. HORSLEY GANTT, M.D. 211 pages; 26.5 × 18.5 cm. 1944. Paul B. Hoeber, Inc., New York City. Price, $4.50.

Lippincott's Quick Reference Book for Medicine and Surgery. Twelfth Edition. By GEORGE E. REHBERGER, A.B., M.D. 1,460 pages; 27 × 18.5 cm. 1944. J. B. Lippincott Co., Philadelphia. Price, $15.00.

Fertility in Women. By SAMUEL L. SIEGLER, M.D., F.A.C.S. 450 pages; 23.5 × 16 cm. 1944. J. B. Lippincott Co., Philadelphia. Price, $4.50.

Fertility in Men. By ROBERT SHERMAN HOTCHKISS, B.S., M.D. 216 pages; 23.5 × 16 cm. 1944. J. B. Lippincott Co., Philadelphia. Price, $3.50. (The two above-mentioned books, in slip case, $8.00.)

Hypertension and Hypertensive Disease. By WILLIAM GOLDRING, M.D., and HERBERT CHASIS, M.D. 253 pages; 24 × 16 cm. 1944. The Commonwealth Fund, New York City. Price, $3.50.

The Argasidae of North America, Central America and Cuba. By R. A. COOLEY and GLEN M. KOHLS. (The American Midland Naturalist. Monograph No. 1. Edited by Theodor Just.) 152 pages; 23.5 × 16 cm. 1944. University of Notre Dame, Notre Dame, Indiana. Price, $2.00.

Textbook of Gynecology. 2nd Edition. By EMIL NOVAK, M.D., F.A.C.S. 708 pages; 16.5 × 24 cm. Williams and Wilkins Co., Baltimore. Price, $8.00.

COLLEGE NEWS NOTES

Additional A.C.P. Members in the Armed Forces

Previously reported in the News Notes section of this journal were the names of 1,706 Fellows and Associates of the College on active military duty. The following additional members have since reported for active duty, bringing the total to 1,714.

Abraham M. Balter
Karl W. Brimmer
William E. Kendall
Frederick W. S. Modern
George W. Pedigo, Jr.
Joseph G. Rushton
Ralph F. Schneider
Walter D. Westinghouse

Lt. Col. Charles H. A. Walton, RCAMC, F.A.C.P., who has served overseas with the Canadian Army, has received an honorable discharge, and will engage again in private practice in Winnipeg, Manitoba.

New Life Members of the College

The following Fellows, listed in the order of subscription, have become Life Members of the College:

Lt. Col. Conrad Acton, (MC), AUS, Overseas
Dr. Robert B. Sanderson, South Bend, Ind.
Dr. Karl Rothschild, New Brunswick, N. J.
Dr. Harold Wentworth Stevens, Marion, Mass.

Gifts to the College Library

The following gifts are gratefully accepted:

Book

Dr. James H. Hutton, F.A.C.P., Chicago, Ill.—"Endocrinology, A Brief Review for Physicians."

Reprints

Edward L. Bortz, F.A.C.P., Captain, (MC), USNR—1 reprint.
Glenn E. Drewyer (Associate), Lieutenant Commander, (MC), USNR—1 reprint.
Dr. Max Millman (Associate), Springfield, Mass.—1 reprint.
Michael Peters (Associate), Lieutenant. (MC), AUS—1 reprint.
Dr. William B. Rawls, F.A.C.P., New York, N. Y.—1 reprint.
G. F. Schmitt, F.A.C.P., Lieutenant, (MC), USNR—1 reprint.
Dr. Maurice S. Segal, F.A.C.P., Boston, Mass.—1 reprint.
Dr. James W. Vernon, F.A.C.P., Morganton, N. C.—2 reprints.
Dr. Salvador Zubiran, F.A.C.P., Mexico City, D. F.—"The New Hospitals of Mexico."
Dr. Leopoldo Herraiz Ballestero, Buenos Aires, Argentina—13 reprints.

A.C.P. Regional Meeting Held at Vancouver, B.C.

Under the direction of an Executive Committee consisting of Dr. George F. Strong, F.A.C.P., Vancouver, Regent and General Chairman, Dr. John W. Scott, F.A.C.P., Edmonton, Governor for the Southwest Provinces of Canada, Dr. Samuel M. Poindexter, F.A.C.P., Boise, Governor for Idaho, Dr. Homer P. Rush, F.A.C.P., Portland, Governor for Oregon, and Dr. Edwin G. Bannick, F.A.C.P., Seattle, Acting Governor for Washington, a Regional Meeting of the American College of Physicians was held at Vancouver September 14–15, embracing Alberta, British Columbia, Manitoba, Saskatchewan, Idaho, Oregon and Washington. Medical officers of the Armed Forces of Canada and the United States in that territory were invited as guests. Dr. David P. Barr, President-Elect of the College, and Commander Corydon M. Wassell, (MC), USNR, the central character represented in the recent moving picture, "The Story of Dr. Wassell," were the chief speakers at the dinner meeting on September 14. Captain Charles E. Watts, (MC), USNR, F.A.C.P., of Seattle, who has served with the Navy in the Pacific, as the chief representative of the Medical Department of the United States Navy, presented two papers on the scientific program, as did also Lt. Col. Roy H. Turner, (MC), USA, Chief of the Communicable Disease Treatment Branch of the Division of Medicine, Office of the Surgeon General, Washington, D. C. Other information and details concerning this meeting will be published later.

A.C.P. Regional Meeting to Be Held in New York City

On October 20, the first Regional Meeting of the American College of Physicians for Eastern New York will be held in New York City under the general chairmanship of Dr. Asa L. Lincoln, F.A.C.P., College Governor for that district. The morning program will be devoted to inspection of scientific exhibits and displays of motion pictures in connection with the Seventeenth Graduate Fortnight of the New York Academy of Medicine. The afternoon program will be devoted to scientific presentations in the amphitheater of the New York Hospital. From six to eight in the evening, there will be a cocktail party and dinner at the Waldorf-Astoria Hotel. In the evening, members of the College will be the guests of the New York Academy of Medicine at a panel discussion on the evaluation of sulfa drugs and penicillin. Dr. David P. Barr, F.A.C.P., will be the leader of the panel, and Drs. René J. DuBos, Colin M. MacLeod, John F. Mahoney, Frank J. Meleney and William S. Tillett will assist.

Fellows and Associates of the College and members of the Medical Corps of the Armed Forces are cordially invited. Programs will be ready for mailing three weeks in advance of the meeting.

Other A.C.P. Regional Meetings Being Planned

A New England Regional Meeting of the American College of Physicians for 1945 will be held early in the New Year at Hartford, Conn., under the general chairmanship of Dr. Charles H. Turkington, F.A.C.P., Governor for Connecticut, and with the assistance of the other New England Governors, including Dr. William B. Breed, Boston, Mass., Dr. Alexander M. Burgess, Providence, R. I., Dr. Harry T. French, Hanover, N. H., Dr. Richard S. Hawkes, Portland, Maine, and Dr. Ellsworth L. Amidon, Burlington, Vt.

Plans are being made for a Regional Meeting of the College for the territory embracing South Carolina, Georgia, Alabama and Florida, at Charleston, S. C., under the general chairmanship of Dr. Kenneth M. Lynch, F.A.C.P., Governor for that State. The date has not yet been determined upon.

A Regional Meeting of the College for Oklahoma and surrounding States is planned at Oklahoma City, February 22, 1945, under the general chairmanship of Dr. Lea A. Riely, F.A.C.P., Governor for Oklahoma. Program is being prepared and will be published in these columns later.

Pan American League Against Rheumatism

The Pan American League Against Rheumatism has been recently organized with the following officers:

Dr. Ralph Pemberton, F.A.C.P., Philadelphia, Pa., *President.*
Dr. Anibal Ruiz Moreno, Argentina, *Vice President.*
Dr. Loring T. Swaim, Boston, Mass., *Secretary.*
Dr. Fernando Herrera Ramos, Uruguay, *Treasurer.*

The countries participating in this League thus far include Argentina, Brazil, Canada, Chile, United States of America, Mexico, Paraguay, Peru and Uruguay. The American Rheumatism Association has appointed Dr. Philip S. Hench, F.A.C.P., as Titulate and Dr. Richard H. Freyberg (Associate), as Alternate to the Central Committee of the League; to the Regional Northern Hemisphere Committee, Colonel Walter Bauer, F.A.C.P., as Titulate and Dr. Donald F. Hill, F.A.C.P., as Alternate.

New Officers of the American Board of Internal Medicine

Dr. Reginald Fitz, F.A.C.P., Boston, has been elected Chairman, Dr. G. Gill Richards, F.A.C.P., Salt Lake City, Vice Chairman, Dr. James J. Waring, F.A.C.P., Denver, Secretary-Treasurer; Dr. William A. Werrell of Madison, Wis., continues as Assistant Secretary-Treasurer.

The American people contributed an all-time record of $10,973,491 to the 1944 Fund-Raising Appeal of the National Foundation for Infantile Paralysis, almost doubling the former record of $5,527,590, set in 1943.

The Foundation is making large contributions to combat the recent outbreaks of infantile paralysis in various parts of the United States, including North Carolina, Kentucky, New York, Louisiana, Pennsylvania, Virginia, Ohio, Tennessee, Michigan, Maryland, Mississippi and Indiana.

Dr. John A. Toomey, F.A.C.P., Cleveland, Ohio, has recently been named Acting Director of Babies and Childrens Hospital and of the Pediatrics Department at Western Reserve University Medical School. Dr. Henry J. Gerstenberger has been granted a leave of absence to devote the remaining years of his active career as Professor of Pediatrics, to publishing results of his extended researches and to do other writing.

International College of Surgeons Meeting

The Ninth Annual Assembly of the International Chapter of the College of Surgeons will be held in Philadelphia, October 3–5, 1944, and the program will be devoted to War, Rehabilitation and Civilian Surgery. Two of the addresses of welcome will be delivered by Fellows of the American College of Physicians, Dr. Rufus S. Reeves, Director of Public Health of Philadelphia, and Dr. Charles R.

Brown, President of Philadelphia County Medical Society. On the evening designated as "Service Night," the guest speakers will be Vice Admiral Ross T. McIntire, F.A.C.P., Surgeon General of the United States Navy, Major General George F. Lull, F.A.C.P., Deputy Surgeon General of the United States Army, Captain Joel J. White, F.A.C.P., of the United States Navy and Dr. Charles M. Griffith, F.A.C.P., Medical Director of the United States Veterans Administration.

7,000 Wounded Flown from Invasion Front in Three Weeks by A.A.F.

Employing new technic to save life as fast as modern war contrives to destroy, the Army Air Forces Medical Corps has pressed into service a new "flying jeep" type of airplane to rush wounded Allied soldiers from the French invasion front to hospitals removed from the scene of battle. More than 7,000 casualties were evacuated by air during the first three weeks following the Normandy invasion, according to Major General David N. W. Grant, Air Surgeon, United States Army Air Forces. General Grant further disclosed that more than 250,000 sick and wounded, American and Allied, have been carried out of battle areas by military air craft since Pearl Harbor. This number is being enlarged, all over the world, at the rate of 1,000 patients a day, he reported.

American People Contributed Liberally to Russian War Relief

During the first six months of 1944, the American people have contributed $13,715,070.99 in cash and contributions in kind to Russian War Relief, more than doubling the amount contributed in the first half of 1943, and more than three million dollars in excess of the quota set for the period covered. All supplies shipped to the Soviet Union carry a label with the American flag on it, and contributions in kind are accompanied by a personal message to the Russian recipient from the original American donor. Nearly nine and a half million dollars worth of goods have been consigned to the Soviet Union since January 1, and an additional four and a half million dollars worth is being processed and packed in Russian War Relief's two major warehouses in New York City, and Portland, Oregon, for immediate shipment.

Handy Index to Hormone Therapy

The Medical Research Division of the Schering Corporation, Bloomfield, N. J., will furnish free, upon request, a completely modernized version, "Handy Index to Hormone Therapy," a useful compilation of data in the hormone field, covering indications, pathogenesis, therapy, rationale and dosage.

Tenth Annual Meeting, Mississippi Valley Medical Society

The Tenth Annual Meeting of the Mississippi Valley Medical Society will be held at the Pere Marquette Hotel, Peoria, Ill., September 27–28. The program will be of a practical nature and will feature bedside medicine. Detailed program may be obtained from the Secretary, Dr. Harold Swanberg, F.A.C.P., W. C. U. Building, Quincy, Ill.

A symposium on the Heart and Circulation will be held at the Louisiana State University School of Medicine, 1542 Tulane Ave., New Orleans, October 25–27, 1944. Dr. Frank N. Wilson, F.A.C.P., Professor of Medicine at the University of Michigan Medical School, will speak on electrocardiography; Dr. Maurice Visscher, of the University of Minnesota, will discuss cardiac efficiency and metabolism; Dr. Isaac Starr, of the University of Pennsylvania School of Medicine, will discuss the ballistocardiograph. Other speakers are from Tulane University and Louisiana State University. No fee will be charged; all who are interested are cordially invited to attend.

News from the Office of the Surgeon General, U. S. Army

Completing his work on his fifth anniversary as Chief of the Professional Service of the Office of the Surgeon General, Brigadier General Charles C. Hillman, F.A.C.P., left Washington, August 7, to take up his new position on or about August 20, as the Commanding General of the Letterman General Hospital, San Francisco. This institution, containing 2500 beds, is the principal debarkation hospital for casualties from the Pacific area. Major General Norman T. Kirk, Surgeon General of the U. S. Army, commenting on General Hillman's transfer said, in part, "General Hillman's assignment as the Commanding General of this important hospital on the West Coast illustrates the Army's concern with the care of sick and wounded soldiers. It is of paramount importance that such work be carried out under the direction of a medical man of wide experience and sound judgment. He has ably directed our Professional Service, being mainly responsible for the initiation of the blood plasma program, resulting in saving the lives of thousands of American soldiers; under his direction was organized the first x-ray examination of all Army inductees, with a lowering of the incidence of tuberculosis among military personnel to less than one-tenth that in World War I. It is a happy coincidence that the qualities of administrative ability and sound medical judgment are thus combined in one man."

General Hillman obtained his doctorate of medicine from Rush Medical College in 1911, after graduating from the University of Arkansas. Following his internship in the Cook County Hospital, Chicago, he entered the Army Medical Corps in 1912. His assignments have been largely professional in character in important medical centers of the Army. His service has included several years at tropical stations during the years of peace and inspection of medical services in overseas theaters in the current emergency. In the autumn of 1943 he visited Brazil as the official guest of the Brazilian Government; and was later decorated in recognition of the assistance that he rendered the Medical Service of the Brazilian Army.

Major General Norman T. Kirk, Surgeon General of the U. S. Army, returned on July 21 from visits to the Italian and Normandy battle fronts where he inspected medical facilities including those at battalion aid stations, as well as Army hospitals in England during his twenty-day trip.

With the advances of Allied forces into former enemy-held territory, the responsibility of the Army for the health of civil populations in occupied and liberated countries in both Europe and the Far East has already been and will be further increased. In meeting this responsibility, The Surgeon General is making use of the advice of outstanding civilian experts in public health. Among those recently appointed as consultants to The Surgeon General, U. S. Army, in matters pertaining to preventive medicine and public health are:

Dr. Claude E. Forkner, F.A.C.P., Director, China Medical Board. Dr. Forkner has recently returned from a year's assignment in China where he was advisor to the Committee on Medical Education of the Ministry of Education of China. While in

China, he served also as Professor of Medicine at the National Central University and the West China Union University, Chengtu, China.

Dr. George K. Strode, Director, International Health Division of the Rockefeller Foundation.

Dr. C–E. A. Winslow, Lauder Professor of Public Health, Yale University School of Medicine, and Editor, Journal of the American Public Health Association.

Dr. Ernest L. Stebbins, Commissioner of Health, New York City.

Plans Shaped for Army Medical History

Representatives of the professional and administrative services of the Office of the Surgeon General, met July 26, 1944, to discuss plans and to make progress reports on the medical history of the war. Colonel Albert G. Love, who was a member of the editorial staff that published the history of the Medical Department of the United States Army in World War I, has been directing the work since August, 1941 on the history of the present war. Marked progress is being made in assembling information from medical installations in this country and overseas. Editors have been selected for the volumes on the medical specialties and the administrative phases of the medical service. In addition to the research and editorial work to be done in the Office of the Surgeon General, historical activities will be carried forward by officers assigned to headquarters of overseas theaters. They will secure first-hand reports of the over-all medical services, particularly those rendered under combat conditions including evacuation of the wounded, the flow of supplies, and other problems. Officers in overseas theaters who have had extensive experience with medical and surgical problems peculiar to this war are being asked to record their observations for the history.

Medical histories were published by the Office of the Surgeon General following the Civil War and World War I. The volumes have done much to perpetuate and disseminate professional, administrative and organizational medical advances developed under the impetus of war. British authorities are carrying forward a similar plan.

Mason General Hospital Dedicated

Surgeon General Norman T. Kirk dedicated the Mason General Hospital, Brentwood, Long Island, New York, June 22, 1944. A special school for training medical officers in neuropsychiatry has been located at this institution. Colonel Cleve C. Odom, F.A.C.P., is the Commanding Officer of the hospital.

Colonel Wakeman Honored Posthumously

Colonel Frank B. Wakeman, F.A.C.P., was awarded the Legion of Merit posthumously for his meritorious work in connection with the training program of the Army Medical Department. The citation reads—

"For exceptionally meritorious conduct in the performance of outstanding services from July, 1940, to March, 1944. Colonel Wakeman, with rare foresight, initiative, and organizing ability, laid the groundwork for the necessary expansion in all phases of Medical Department training, placing in operation replacement training centers, service schools for officers, Medical Department Enlisted Technicians Schools, and an Officer Candidate School, long before the entry of the United States into the

war. As a result of his insight into medical requirements and the execution of plans, the Medical Department was able to expand greatly its training activities following December 7, 1941, and also, because of training already given, to render an efficient medical service to the Army during the very rapid expansion that followed the declaration of war. Colonel Wakeman's unusual foresight, aggressive execution of approved plans, and selfless devotion to the best interests of the Army and the Medical Department are in the highest traditions of the service."

Colonel Wakeman was awarded the Henry Welcome Prize in 1938 for his thesis on an immunizing antigen of the typhoid bacillus. He died in March of 1944 of a coronary occlusion while attending a conference at Fort Monmouth, N. J.

Colonel Flickinger Returns from Burma

Colonel Don Flickinger (Associate), formerly Wing Surgeon of the A.A.F. Air Transport Command, India-China wing, recently returned to the United States for a new assignment. Colonel Flickinger, it will be remembered, parachuted into the Burma jungle in August, 1943, to bring aid to the victims of a plane crash. He brings glowing reports of the volume and success of air evacuation of casualties from the China-Burma fighting front.

Colonel Flickinger was awarded the Legion of Merit for working upon methods of sighting pilots forced down at sea. He also holds the Distinguished Flying Cross, Soldier's Air Medal and a Presidential unit citation.

Major William O. Benenson (Associate), formerly of Napanoch, N. Y., has been advanced to Chief of the Medical Service of the ASF Regional Hospital, Fort Benning, Ga., and Captain Michael Peters (Associate), formerly of Telford, Pa., has been advanced to the Assistant Chief of the Medical Service of the same hospital.

Major William J. Mitchell (Associate), formerly of Alhambra, Calif., has been named Commanding Officer of the Station Hospital at Fort Douglas, Utah.

Dr. Harold R. Carter (Associate), Denver, Colo., addressed the Larimer Medical Society, Berthoud, Colo., June 6, 1944, on "Present Day Trends in the Diagnosis and Treatment of the Psychoneuroses."

Dr. Paul F. Whitaker, F.A.C.P., Governor for North Carolina, was installed as President of the Medical Society of the State of North Carolina at the annual meeting at Pinehurst, N. C., May 1. Dr. William H. Smith, Goldsboro, N. C., was elected Vice-President.

Lt. Col. Thomas Fitz-Hugh, Jr., F.A.C.P., formerly of Philadelphia, was promoted to the grade of Colonel on July 25, 1944.

Dr. Louis H. Bauer, Hempstead, N. Y., has been elected a member of the Board of Trustees of the American Medical Association.

Colonel Garfield G. Duncan, F.A.C.P., formerly Associate Professor of Medicine, Jefferson Medical College, and Chief of the Medical Service of the Pennsylvania Hospital, Philadelphia, has been awarded the Legion of Merit by command of General MacArthur for his work on malarial control and atabrine studies.

Dr. William Earl Clark, F.A.C.P., and Dr. William M. Ballinger, F.A.C.P., are President-Elect and First Vice President, respectively, of the Medical Society of the District of Columbia.

Dr. Edward Sterling Nichol, F.A.C.P., Miami, has been elected President of the American Therapeutic Society, and Dr. Oscar B. Hunter, F.A.C.P., Washington, has been elected Secretary.

Dr. R. D. Thompson, F.A.C.P., Medical Director of the State Tuberculosis Sanatorium, Orlando, Fla., has been elected President of the Southern Conference on Tuberculosis.

Dr. William H. Gillentine, F.A.C.P., has been made President-Elect of the New Orleans Graduate Medical Assembly for 1944–1945.

Dr. J. W. Finch, F.A.C.P., Hobart, Okla., was recently appointed Chairman of Procurement and Assignment of Kiowa County.

Dr. S. E. Thompson, F.A.C.P., Kerrville, Dr. Robert G. McCorkle, F.A.C.P., San Antonio, and Dr. H. F. Carman, F.A.C.P., Dallas, have been elected President, First Vice President and Second Vice President, respectively, of the Texas Chapter, American College of Chest Physicians.

Dr. E. V. DePew, F.A.C.P., San Antonio, Tex., has succeeded Dr. H. J. Schattenberg, F.A.C.P., as a member of the San Antonio Board of Health. Dr. Schattenberg resigned to resume his place as Consultant to the State Board of Health.

Dr. R. Finley Gayle, F.A.C.P., Richmond, was recently elected a member of the National Committee for Mental Hygiene.

Dr. John T. O'Mara, F.A.C.P., Baltimore, has been reëlected Secretary-Treasurer of the Board of Medical Examiners of Maryland.

Dr. Rollin H. Stevens, F.A.C.P., Detroit, is President of the Detroit Institute of Cancer Research, which was established in 1941. The Institute has purchased from the Detroit Edison Company the Detroit Edison Club property, will remodel the two buildings as a temporary cancer research laboratory and hopes to coöperate closely in cancer research with the proposed Medical Science Center of Wayne University.

Dr. Alexander H. Stewart, F.A.C.P., Harrisburg, Dr. Stanley P. Reimann, F.A.C.P., Philadelphia, and Dr. William H. Perkins, F.A.C.P., Philadelphia, were speakers on the program of a conference on health and human relations held under the auspices of the Pennsylvania Department of Health at State College, Pa., July 18–19. The program dealt chiefly with sex education.

Dr. John F. Kenney, F.A.C.P., Pawtucket, has been made President-Elect of the Rhode Island Medical Society. Dr. Elihu S. Wing, F.A.C.P., Providence, is the present incumbent.

Dr. Chester S. Keefer, F.A.C.P., Boston, has been appointed the Medical Administrative Officer of the Committee on Medical Research. Several new divisions have been established. Dr. E. Cowles Andrus, F.A.C.P., Baltimore, is Chief of the Division of Medicine, and Dr. Joseph T. Wearn, F.A.C.P., Cleveland, is Chief of the Division of Physiology. The headquarters office in Washington, D. C., is at 2101 Constitution Ave., N. W.

Dr. Roy L. Leak, F.A.C.P., recently resigned as Superintendent of the Connecticut State Hospital, Middletown, after holding the position since April, 1922.

Dr. Theodore G. Klumpp, F.A.C.P., President of the Winthrop Chemical Company, has announced the establishment of a division of product development which will explore the post-war commercial potentialities of products being supplied by this Company to the Armed Forces as well as new products developed in the Company's laboratories.

Dr. Klumpp is also Chairman of the Advisory Committee of Physicians on Drug Exhibits of the New York Academy of Medicine. The Academy has set up plans for a continuous non-commercial drug exhibit at its headquarters. An adequately qualified person will be in charge to answer questions and explain new drugs. Pamphlets and literature will be furnished, giving the research and clinical usage and describing the products.

Dr. Tasker Howard, F.A.C.P., after being connected with the Long Island College of Medicine for some 34 years, has retired as Professor and Executive Officer of the Department of Medicine.

Dr. William Dock, Professor of Medicine at the University of Southern California, Los Angeles, has accepted the position, on a full-time basis, at the Long Island College of Medicine.

Dr. Alexander M. Burgess, F.A.C.P., Providence, R. I., has been appointed Professor of Health and Hygiene in a newly created department of medical science at Brown University. The new department is intended to assist the University in assuming a larger responsibility for the general education of its students in matters of health.

Dr. Roy L. Sexton (Associate), Washington, D. C., is a member of a three-man medical mission sent to the Pribilof Islands in the Bering Sea to assist local medical personnel in the examination of 400 Aleut natives, recently repatriated after two years in southeastern Alaska.

The Chicago Medical Society has been sponsoring a series of popular health talks, given at the Museum of Science and Industry, Chicago. The first lecture, "Facts about Cancer," was delivered by Dr. Josiah J. Moore, F.A.C.P., President of the Chicago Medical Society and Treasurer of the American Medical Association. Dr. Samuel M. Feinberg, F.A.C.P., gave the lecture on August 2, "Allergy, Facts and Fiction"; Dr. James H. Hutton, F.A.C.P., August 9, "Your Glands and What They Do to and for You"; and Dr. Andrew C. Ivy, F.A.C.P., August 23, "Aviation Medicine."

Dr. Oliver Perry Kimball, F.A.C.P., Cleveland, and Dr. Margaret Bell, F.A.C.P., Ann Arbor, were among physicians cited by the University of Chicago on June 10 as "useful citizens." Dr. Kimball was largely responsible for the collection of funds from Cleveland alumni to establish a local scholarship at the University, and Dr. Bell is Professor of Hygiene and Physical Education, Chairman of the Physical Education for Women and physician in the Health Service of the University of Michigan.

Dr. Edward A. Strecker, F.A.C.P., Philadelphia, delivered an address on "The Contribution of Psychiatry to Democratic Morale," at the 100th anniversary of the Butler Hospital, Providence, R. I., May 10. This is the oldest hospital in the State.

Brigadier General John M. Willis, F.A.C.P., Surgeon of the 9th Service Command, U. S. Army, Fort Douglas, Utah, addressed the Utah State Medical Association's annual meeting at Salt Lake City, August 25–26, on "Administration and Hospitalization in a Service Command." Lt. Col. Lauren H. Smith, F.A.C.P., addressed the same meeting on "Treatment in War Psychiatry."

Dr. Richard H. Freyberg (Associate), heretofore Assistant Professor of Internal Medicine at the University of Michigan Medical School, Ann Arbor, has accepted an appointment as Chief of the Department of Medicine and Pediatrics at the Hospital of Special Surgery, New York City, effective September 1, 1944.

Dr. Floyd L. Rogers, F.A.C.P., Lincoln, Nebraska, is President of the Nebraska State Medical Association. Dr. William J. Reeder (Associate), Cedar Rapids, is a Vice President.

Dr. E. Roland Snader, Jr., F.A.C.P., Philadelphia, was elected to the Council of the American Diabetes Association at the recent June meeting in Chicago. Dr. Edward S. Dillon, F.A.C.P., Philadelphia, was elected Second Vice President at the same meeting.

War-Time Graduate Medical Meetings

Region No. 1 (Maine, New Hampshire, Vermont, Massachusetts)—Dr. C. S. Keefer, Chairman; Dr. M. C. Sosman, Dr. A. W. Allen.

Region No. 2 (Connecticut, Rhode Island)—Dr. S. B. Weld, Chairman; Dr. C. Barker, Dr. A. M. Burgess.

Station Hospital, Dow Field, Bangor, Maine

September 21 Burns and Reconstruction Surgery

Dispensary, U. S. Naval Air Station, Brunswick, Maine

September 21 The Psychoneuroses and Their Management

Station Hospital, Fort Williams, Portland, Maine

September 21 Cardiac Neuroses, Cardiac Emergencies, Cardiac Rehabilitation—Drs. Samuel H. Proger and T. Duckett Jones

Station Hospital, Presque Isle, Maine

September 21 The Use of Penicillin and the Sulfa Drugs—Dr. Charles A. Janeway

Dispensary, U. S. Naval Construction Training Center, Quoddy Village, Maine

September 21 The Pneumonias and Other Respiratory Infections—Dr. Cutting B. Favour

Station Hospital, Grenier Field, Manchester, New Hampshire

September 20 Acute Abdominal Emergencies

U. S. Naval Hospital, Portsmouth, New Hampshire

September 21 Chest and Abdominal Injuries

Station Hospital, Fort Banks, Boston, Massachusetts

September 21 The Skin—Dr. Francis M. Thurmon

U. S. Naval Hospital, Chelsea, Massachusetts

September 21 Acute Infections of the Central Nervous System—Dr. Derek E. Denny-Brown

Station Hospital, Fort Devens, Massachusetts

September 21 Stomach, Biliary Tract, Intestinal Disorders—Drs. J. Howard Means, Robert R. Linton and Laurence L. Robbins

Station Hospital, Camp Edwards, Massachusetts

September 21 Tropical Diseases, to Include Malaria and Other Insect-Borne Diseases

Cushing General Hospital, Framingham, Massachusetts

September 21 Peripheral Vascular Disease—Dr. E. Everett O'Neil

Station Hospital, Camp Myles Standish, Taunton, Massachusetts

September 21 Contagious Diseases and Complications—Dr. Edwin H. Place

U. S. Marine Hospital, Brighton, Massachusetts

September 21 Blood Dyscrasias and Transfusions—Dr. Louis K. Diamond

Station Hospital, Westover Field, Chicopee Falls, Massachusetts

September 21 Pilonidal Sinus and Common Diseases of the Anus and Rectum—Dr. E. Parker Hayden

Dispensary, U. S. Naval Construction Training Center, Davisville, Rhode Island

September 21 Diarrheal Diseases—Drs. Ralph E. Wheeler and Francis C. McDonald

U. S. Naval Hospital, Newport, Rhode Island

September 21 Cardiac Neuroses, Cardiac Emergencies, Cardiac Rehabilitation—Drs. Paul D. White and Mandel E. Cohen

Station Hospital, Bradley Field, Windsor Locks, Connecticut

September 21 Joint Injuries—Dr. John H. T. Sweet, Jr. and associates

Air Corps Station Hospital, New Haven, Connecticut

September 21 Fractures of Extremities—Dr. Frank S. Jones

Station Hospital, Fort H. G. Wright, Fishers Island, New York

September 21 Head, Spine and Nerve Injuries

REGION No. 10 (Kentucky, Tennessee)—Dr. E. L. Henderson, Chairman; Dr. C. W. Dowden, Dr. H. H. Shoulders.

Combined War-Time Graduate Medical Meeting and Kentucky State Medical Association Meeting, Lexington, Kentucky

September 19 Chemotherapeutics in Pediatrics—Dr. John A. Toomey
Chemotherapy (Penicillin)
Civilian Medical Essayist—Dr. Donald G. Anderson
Symposium (Military Essayists)
Medical Aspects—Brigadier General Hugh Morgan
Surgical Aspects—Colonel B. N. Carter
Venereal Disease Treatment—Lieutenant Colonel Thomas Sternberg
Oration in Medicine—Dr. Frederick G. Speidel
Psychosomatic Medicine—Dr. Maurice Levine
Industrial Health Hazards—Colonel Anthony Lanza
Current Problems in Aviation Medicine—Major General David N. W. Grant
The Present Status of Pain Relief in Labor—Dr. Frederick H. Falls
Cardiovascular Diseases—Dr. William D. Stroud
(Evening Program)
President's Address—Dr. Oscar O. Miller
Accelerated Medicine Today and Tomorrow—Dr. Roger I. Lee
Address—Dr. Edward Henry Cary

September 20 Symposium on Tropical Diseases
Epidemiology—Dr. R. E. Dyer
Medical Aspects of Tropical Diseases—Captain Alphonse McMahon
The New Weapons for Control of Insect-borne Diseases—Brigadier General James S. Simmons
The Surgical Aspects of the Chronic Dyspepsias—Dr. Irvin Abell
Oration in Surgery—Dr. J. Farra Van Meter
Arthritis—Dr. Ralph Pemberton
Address—Brigadier General Fred W. Rankin
Nutrition—Its Relation to Deficiency Diseases—Colonel John D. Youmans

REGION No. 14 (Indiana, Illinois, Wisconsin)—Dr. W. O. Thompson, Chairman; Dr. N. C. Gilbert, Dr. W. H. Cole.

Mayo General Hospital, Galesburg, Illinois

September 20 Diseases of the Kidneys, Uro-genital Tract
Diseases of the Kidneys—Edema
Surgical Considerations

October 4 Blood Dyscrasias
Acute and Chronic

Camp Ellis, Illinois

September 20 Conditions Affecting Glucose Metabolism
Endocrine—Pituitary—Thyroid—Adrenal—Pancreatic
Renal, Alimentary, Hepatic. Diff. Diagnosis and Treatment

October 4 Orthopedic Problems of General Interest
Low Back Pain—Foot and Knee Strain—March Fracture, etc.

Camp McCoy, Wisconsin

September 20 Diseases of the Intestinal Tract
Regional Ileitis, Colitis, Diverticulitis. Diagnosis and Treatment.
Dysentery—Army and Bacillary
Malignancies

October 4 Symposium on Organic Neurology
Central and Peripheral

Camp Grant, Illinois

September 20 Dermatological Diseases
Clinic with Presentation of Cases and Slides. Diagnosis and Treatment
The Less Common Venereal Diseases
Lymphogranuloma Venereum, Granuloma Inguinale, Chancroid, Yaws

October 4 Psychiatry—Psychoneuroses—Neurocirculatory Asthenia—Malingering, etc.

Truax Field, Wisconsin

September 20 Malignancies in the Army Age Group
Melanomata
Teratomata
Lymphoblastomata

October 4 Endocrinology
Addison's Disease, Adrenal Cortex in Shock, Parathyroid Tetany, Traumatic Hypogonadism, Hypothyroidism, Hyperthyroidism, Post-Traumatic Pituitary Syndrosis

Chanute Field, Rantoul, Illinois

September 20 Chemotherapy—Present Status
Sulfonamides
Penicillin
Gramicidin

October 4 Gall Bladder and Liver Disease
Mechanism of Liver Function
Diagnosis and Medical Treatment of Liver and Gall Bladder Disease
Surgical Pathology and Treatment

(The names of those speakers which do not appear on the above schedule are to be announced.)

American Clinical and Climatological Association

Francis M. Rackemann, M.D., *Secretary*

263 Beacon Street, Boston 16, Mass

August 11, 1944

To the Members of the Council:

In re: The Next Meeting

Our President, Dr. C. Sidney Burwell, and I have been considering the matter carefully, and we submit to you our thoughts with the request that you send us your approval and/or your comments in the near future.

Last May in Atlantic City, several members discussed the next meeting informally and decided to postpone decision "until we could see how the war was going." The war is going well but it is not won—our troubles are not yet over and they are not likely to be over for many months to come. All our young members are in Service and as for the older members it is most impressive to see the important positions which they hold and the heavy responsibilities which they carry. Whereas many men would try hard to come to a meeting, the trip would entail a large effort and the men would not stay long. Incidentally, transportation is still difficult and still restricted.

To us, therefore, it seems wisest to postpone the meeting for one more year and to hope that in October 1945 we can have the most interesting, the biggest and the best meeting which the "Climatological" has ever held. What do you say?

With kind regards,

Yours sincerely

Francis M. Rackemann
Secretary pro tem

Lieut. Colonel Martin A. Compton

Appointed to Surgeon General Board

Lieut. Colonel Martin A. Compton, M.C., has been appointed as a member of a board of the Office of The Surgeon General, the purpose of which is to prepare, develop and implement the medical portion of the War Department's program for aid to civilian populations in liberated countries. This board was established in June, 1943.

Colonel Compton was born in Palmyra, Illinois, in 1913, attended Bradley Polytechnic Institute and Washington University School of Medicine (St. Louis) and received his medical degree from the Washington University School of Medicine (St. Louis). In 1938 he was appointed First Lieutenant in the Medical Corps and attained a Captaincy two years later. In 1942 he was promoted to the grade of Major and became Lieut. Colonel in the Fall of 1943.

Colonel Martin A. Compton has been associated with and Chief of the Requirements Branch in the Office of The Surgeon General since 1941.

Lieut. Colonel Robert James Wilson, M.C., Dies

The War Department has announced the death of Lieut. Colonel Robert James Wilson, M.C., staff officer of the United States Army Bruns General Hospital, Santa Fe, New Mexico, which occurred on July 11. Grave-side services were held at Arlington Cemetery July 20; he was accorded a military funeral.

Colonel Wilson was born in 1906 in Buffalo. After completing his work at the Kentucky Military Institute he obtained the Bachelor of Arts degree at the University of Maryland in 1927. The University of Buffalo awarded him the degree of Doctor of Medicine in 1931. He received his commission as First Lieutenant in the Medical Corps in June of that year, serving internship at the Walter Reed General Hospital; there he served as assistant chief ward officer in the surgical service. Upon completing this work, he was assigned to the Army and Navy General Hospital at Hot Springs, Arkansas.

He became associated with the Office of The Surgeon General Finance and Supply Division in 1941. Shortly thereafter he was appointed Chief of the Civilian Personnel Division—the position which he held until his assignment to the Bruns General Hospital in 1942.

Army Increases Officer Candidate Quotas for Medical Administrative Corps

An increase in quotas for admission to officer candidate courses leading to commissions in the Medical Administrative Corps of the Army has been announced by the War Department.

Quotas which until recently have been extremely limited have been revised to permit acceptance of 2,000 men within the next eight weeks for 17-week courses. The primary reason for the increase is the need for more officers qualified for administrative duties in the Army Medical Department to free members of the Medical Corps for professional duties.

In recent months only the Medical Administration Corps Officer Candidate School at Camp Barkeley, Texas, has been accepting candidates. Under the new plan, the Officer Candidate School at Carlisle Barracks, Pennsylvania, was re-opened on June 24.

The August Army Medical Bulletin

The following articles appeared in the August issue of the *Army Medical Bulletin:*

Laboratory Aids in Diagnosis of Rocky Mountain Spotted Fever
Colonel Harry Plotz, M.C.
1st. Lieut. Kenneth Wertman, Sn.C.
Captain Reginald L. Reagan, Sn.C.
Plaster of Paris Casts and Splints
Captain J. Vernon Luck, M.C.
Medical Service in the New Georgia Campaign
Nomogram for Computing Morbidity and Mortality Rates
Anesthesia in the Combat Zone
Captain Gerald Shorts, M.C.
Cutaneous Leishmaniasis
Major David Ball, M.C.
Captain Raymond C. Ryan, M.C.
Oral Rehabilitation
Captain R. C. Reichert, D.C.
Herniated Nucleus Pulposus
Major Robert C. L. Robertson, M.C.
Captain William G. Peacher, M.C.
Surgical Problems in the Buna Campaign
Colonel Augustus Thorndike, M.C.

Vaccinia Occurring at Short Intervals
Captain Carl A. Minning, M.C.
Inspection of Fish of the Pacific Northwest
Captain Ernest W. Bloomquist, V.C.
The Diagnosis of Dengue
Major George V. LeRoy, M.C.
Captain Howard A. Lindberg, M.C.
Experimental Use of Penicillin in Treatment of Sulfonamide-Resistant Gonorrhea
Captain Robert J. Murphy, M.C.
Modified Orthopedic Table Constructed in the Field
Captain Victor Mayer, M.C.
Psychoses in the Army
Major Norman Q. Brill, M.C.
Captain Edmund F. Walker, M.C.
Rocky Mountain Spotted Fever
Major Oscar A. Palatucci, M.C.
Major Bruno A. Marangoni, M.C.

Clinical Psychologists Aid Army

Clinical Psychologists commissioned in the Adjutant General's Department are being made available for assignment to the Office of The Surgeon General for the neuropsychiatric sections of named and numbered general and station hospitals of 1,000 beds or more, the War Department announced on July 1, 1944. Requisitions for such officers will be forwarded to the Adjutant General through commanding generals of service commands concerned, or theater commanders when applicable.

Clinical psychologists will be assigned to duty in the neuropsychiatric sections of the hospital to serve under the direction and supervision of the chief of the neuropsychiatric section. Their duties will be to—

a. Aid in the development and administration of the program of counseling designed to prepare convalescent patients for return to military service.

b. Assist in the preparation of clinical records, particularly including those requiring the use and interpretation of special psychological tests as desired by the chief of the neuropsychiatric section.

c. Assist in studies of special psychological problems related to the classification and retraining of neuropsychiatric casualties.

d. Assist in the determination of the appropriate military occupational specialty of men who are designated as ready for assignment, and to advise regarding their assignment to a specific duty or special training.

e. Perform such other professional and administrative duties in the hospital as will best assist the neuropsychiatrist in the accomplishment of the best management and disposition of patients.

Postwar Training of Medical Corps Officers

The Office of The Surgeon General has announced the appointment of a committee to formulate plans for postwar training of Medical Corps Officers who will be separated from the military service at the end of the war. The committee consist of: Brig. General Raymond W. Bliss, Chief of Operations Service—*Chairman;* Brig. General James S. Simmons, Chief of Preventive Medicine Service; Colonel James R. Hudnall, Chief of Personnel Service; Brig. General Fred W. Rankin, Director of Surgery Division; Brig. General Hugh J. Morgan, Director of Medicine Division; Colonel Floyd L. Wergeland, Director of Training Division; Colonel Wil-

liam P. Holbrook, M.C. and Lieut. Colonel R. H. Meiling, M.C., representatives from the Army Air Forces; Colonel R. B. Skinner, M.C. representative from the Army Ground Forces; George B. Darling, M.D., representative from the National Research Council.

George M. Powell Promoted

Colonel George M. Powell, M.C., director of the Special Planning Division, Operations Service in the Office of the Surgeon General, advanced from the grade of Lieut. Colonel on June 15. He was born in Los Animas, Colorado, in 1906 and graduated from the Colorado College in 1927. He obtained his medical degree from the Washington University School of Medicine in St. Louis, Missouri. In 1939 he completed nine months of training in Internal Medicine at the Walter Reed General Hospital.

Colonel Powell served as Cardiologist and later became Chief of the Medical Service at Gorgas Hospital, Ancon, Canal Zone, where he was stationed from the Fall of 1939 to the Fall of 1942. Then he was assigned to the Medical Replacement Center of Camp Joseph T. Robinson in Arkansas, where he performed the duties of Assistant Regional Commander until he became full Regional Commander in 1943.

Decorations for Scrub Typhus Control Service

The Bronze Star Medal has been awarded to an officer of the Sanitary Corps and seven enlisted men of the Medical Department for their services in the prevention of scrub typhus on Goodenough Island in the South Pacific as announced on May 16, 1944 by the Sixth Army Headquarters.

Although these men were fully aware of the danger of contracting scrub typhus, they voluntarily applied themselves to the task of preparing camp sites in order to bring about the rapid and complete control of this disease on Goodenough Island. Their services were rendered during the periods December 27, 1943 to February 7, 1944 and March 13–22, 1944.

U. S. Army Blind Centers

The Army Medical Department has designated two hospitals, namely, Valley Forge General Hospital, Phoenixville, Pa., and Dibble General Hospital, Menlo Park, California as blind centers where all blinded casualties are being sent to receive a preliminary course in social rehabilitation while undergoing any necessary medical or surgical treatment.

On July 21 the first group of blinded casualties was received at Old Farms Convalescent Hospital (Special), Avon, Conn., which has been designated as the blind center where the final phase of social rehabilitation is given. This group has undergone the preliminary training at Valley Forge General hospital, and during their stay at Old Farms will receive more advanced training in social rehabilitation prior to discharge from the Army and transfer to the Veterans Administration.

Major Alexander Pierce Ormond, F.A.C.P. (MC), AUS, Chief of Medical Service of the Station Hospital, Reno Army Air Base, Reno, Nevada and formerly of Akron, Ohio, gave an address before the Nevada Branch of the American Social Hygiene Association in Reno on July 12, 1944. He spoke on "The Army's Venereal Disease Control Program." Dr. Lawrence Parsons, F.A.C.P., president, introduced the speaker.

Governor Bricker Comments on American Medicine

Governor John W. Bricker of Ohio, Republican Vice Presidential candidate, in addressing the Creve Coeur Club at Peoria last February 22 said, among other things, "The American doctors have made eminent progress in caring for the health of our people. Medical organizations and private hospital groups are making substantial progress toward the goal of providing adequate medical and hospital care for all. In view of this record I regard the proposals emanating from this administration for governmental intervention between the doctor and his patient as an undeserved affront to a loyal and admirable profession, and a distinct threat for the future health of our people. It is these meddlesome activities in so many spheres that properly belong to the states or to the people themselves that have led to the multiplicity of government agencies which are unsupervised and uncontrolled, and which it is impossible to supervise or control. Please do not misunderstand me. Government must be responsive to the needs of social progress in every field. It must continue to be. Human welfare means more than good intentions and material help. It must promote education, health and public welfare. But it must leave to individual human beings a full measure of control over their own destiny. Governmental management and regimentation invariably lead to national chaos and disorder."

OBITUARIES

DR. KENNON DUNHAM

Dr. Kennon Dunham, F.A.C.P., Cincinnati, Ohio, died April 27, 1944, of coronary thrombosis; aged 72.

Dr. Dunham was born in Fairfield, Ohio, in 1872, and received his medical degree from Miami Medical College, now the University of Cincinnati College of Medicine, 1894. He later did postgraduate work at Johns Hopkins University School of Medicine, the University of Wisconsin Medical School, in London and in Germany. For many years he was Associate Professor of Medicine and Head of the Department of Tuberculosis at the University of Cincinnati College of Medicine and the Cincinnati General Hospital. At one time he was Attending Physician to the Cincinnati Tuberculosis Sanatorium, and during World War I he served in the Medical Corps of the U. S. Army as Chief of the Laboratory Service at Oteen, N. C.

Dr. Dunham was a former President, 1921, of the Cincinnati Academy of Medicine, a member of the Ohio State Medical Society, the American Roentgen Ray Society, the American Clinical and Climatological Association, the National Tuberculosis Association, Society of Thoracic Surgery, a Fellow of the American Medical Association, and had been a Fellow of the American College of Physicians since 1923. He was the author of many published papers, and a physician who will be sorely missed.

DR. GEORGE McCLINTOCK HUTCHISON

Dr. George McClintock Hutchison, F.A.C.P., Ridgway, Pa., died January 6, 1944, of peritonitis; aged 68.

Dr. Hutchison was born in Brockway, Jefferson County, Pa., November 18, 1875. In 1900 he graduated from the Pennsylvania College of Dental Surgery with the degree of D.D.S., and in 1907 from the Medico-Chirurgical College of Philadelphia with the degree of M.D. In 1927 he completed the course at the University of Pennsylvania Graduate School of Medicine, receiving the degree of Master of Medical Science.

For many years Dr. Hutchison was a practicing physician at Dagus Mines and Kersey, Pa. For the last several years he practiced internal medicine at Ridgway. He was a member of the Staff of the Elk County General Hospital and a Visiting Physician to the Andrew Kaul Memorial Hospital at St. Marys. He was a member of the Elk County Medical Society, the Pennsylvania State Medical Society, a Fellow of the American Medical Association, and had been a Fellow of the American College of Physicians since 1936.

DR. ANGUS MacKAY

Dr. Angus MacKay, M.B., F.A.C.P., Toronto, Ont., Canada, died March 4, 1944, from an acute attack of coronary thrombosis; aged 54.

Dr. MacKay was born in the Township of West Zorra, County of Oxford, and was a direct descendant of the Sutherland MacKays who were pioneers of that section of Ontario. His early education was attained in Embro and the Collegiate Institute of Woodstock. He received his Bachelor of Medicine degree from the University of Toronto in 1916. From that date until 1919, he served with the rank of Captain in the Royal Canadian Army in Canada, England and France. He interned at St. Michael's Hospital and assisted Dr. David Smith, of Stratford, for a time before entering practice in Toronto. He did postgraduate work in New York and in Rochester, Minn.

As a classmate and friend of the late Sir Frederick Banting, F.A.C.P., he was one of the first to study the clinical application of insulin, and was soon recognized as an authority on its use in the treatment of diabetes. He limited his practice to Internal Medicine, and was made a Fellow of the American College of Physicians in 1931.

In the Toronto Western Hospital he had charge of the diabetic patients, but he was a favorite consultant also in other departments of the hospital. His marked common sense and his clinical acumen won the confidence of his colleagues, and his loss is a major calamity to the staff.

Dr. MacKay made valuable contributions to the literature of diabetes and hypertension; he wrote book reviews with discrimination; he served for years on the Publication Committee of the Toronto Academy of Medicine, was a member of its Council from 1938 to 1941 and Chairman of the Section of Pathology, 1930–31, and of the Section of Medicine, 1940–41. His hobbies were fishing, gardening, golf and the reading of historical novels and biographies. He is survived by his wife, Mrs. Edna Catherine Hanna MacKay, a son, Ross Cameron and a daughter, Elizabeth Ann.

—Taken in part from the Bulletin of the
Toronto Academy of Medicine

DR. HOMER WOOLERY

Dr. Homer Woolery (Associate) of Bloomington, Ind., died April 22, at the age of 71 of coronary thrombosis.

Dr. Woolery was born December 24, 1872; received his A.B. degree in 1897 in Indiana University, and his M.D. in 1907 from the same institution. He later pursued postgraduate study at Harvard Medical School. He practiced the specialties of internal medicine and pediatrics in Bloomington for a great many years. He was a member of the Phi Delta Theta and Nu Sigma Nu Fraternities, also a member of the Monroe County Medical Society, the Indiana State Medical Society, the Central States Pediatric Society and the American Academy of Pediatrics. He had been an Associate of the American College of Physicians since 1921 by virtue of former membership in the American Congress on Internal Medicine.

Dr. Woolery was well liked by his fellow practitioners and was well known to the Indiana University student body over a period of many years, for he was a friend to all of them.

ROBERT M. MOORE, M.D., F.A.C.P.,
Governor for Indiana

DR. CHARLES HARTWELL COCKE

Dr. Charles Hartwell Cocke, First Vice-President of The American College of Physicians, died of coronary occlusion on the morning of August 3, 1944. Stricken on July 29, he realized that his illness would probably be fatal and, with his customary thoughtfulness and consideration, he furnished his secretary, Miss Studebaker, with a list of names to be notified in case of his death. Dr. Paul H. Ringer, his physician and devoted friend of thirty-three years standing, was with him at the end.

Dr. Cocke was born in Columbus, Miss., on December 1, 1881, the son of Charles Hartwell Cocke, who was the second President of the Industrial Institute and College, now known as Mississippi College for Women, and Rowena Lockart Hudson Cocke. He received his early education at Episcopal High School, where he was an honor student and leader in school activities. From there he went to the University of Virginia where he graduated with an A.B. degree in 1902. He received his M.D. degree from Cornell University Medical College in 1905, and in 1908 he did postgraduate study at the Allgemeines Krankenhaus, Vienna, and the hospitals of London and Paris. In 1914 he married Miss Amy Grace Plank, of Carlisle, Pa. Their home life was ideal and they lived happily through the years. Mrs. Cocke was with her husband at the end. Many is the visitor who has enjoyed the cordial hospitality of this gracious couple in their home.

Going to Asheville, North Carolina, by reason of impaired health, he remained to become one of that city's most illustrious, useful and respected citizens. He was for many years Attending Physician for the Asheville Mission and Biltmore Hospitals. He was Consulting Physician to the Learline Reaves Sanatorium (Greenville, Tenn.) and Patton Memorial Hospital, Hendersonville, North Carolina. He was co-founder and medical director of the Zephyr Hill Sanatorium, and consultant to most of the hospitals of Asheville.

Dr. Cocke was a Diplomate of the American Board of Internal Medicine; President, 1923, Buncombe County Medical Society; Vice-President, 1927, Medical Society of the State of North Carolina; Vice-President, 1932, Southern Medical Association; Vice-President, 1934, American Clinical and Climatological Association; Vice-President, 1935 and 1936, American College of Chest Physicians; Member of National Tuberculosis Association, Southern Interurban Clinical Club, American Association of the History of Medicine, American Heart Association; Fellow, American Medical Asso-

ciation; Member of the Phi Kappa Psi and Nu Sigma Nu fraternities, and, during World War I, he was Chief Consultant and Secretary of his local Medical Advisory Board.

The Medical Society which he enjoyed and loved most was The American College of Physicians. He became a Fellow of the College in 1928 and was elected to the Board of Governors, representing the State of North Carolina, in 1929. He was Chairman of the Board of Governors from 1925 to 1942 and became Vice-President of the College in 1942; was re-lected in 1944 and held that office at the time of his death. He served on

CHARLES HARTWELL COCKE, Asheville, N. C.
Regent, and Chairman, Board of Governors

numerous committees of the College; he was a member of the Committee on Credentials for many years and on this Committee he performed signal service in connection with the standards of the College. Dr. Cocke loved the College and everything about it and maintained an abiding interest in its welfare and progress. To him, more than any other man, goes the credit for the fine and healthy growth of the College in his adopted State of North Carolina. Giving his enthusiastic support and his recognized talent to the promotion of its welfare from 1928 on, it can be truthfully said that his passing has lost for the College one of its most loyal and valuable members.

Dr. Cocke was author of numerous publications in the best medical journals in the country. Throughout his professional life there were very few years that he did not make at least one contribution to medical literature.

Although medicine was his primary interest, Dr. Cocke, being the good citizen that he was, gave liberally of his time and talent to the civic and church affairs of his home city, Asheville. For years he was a member of the Asheville Chamber of Commerce, serving as Vice-President in 1939 and Director from 1938 to 1940. He served on the Board of Directors of the Asheville Community Chest for three years. A member of the Trinity Episcopal Church, he served as vestryman for twenty years, was a former Senior Warden, Trustee of the Diocese and a member of the Standing Committee of the Diocese for many years. He also served as President of the Men's Bible Class of his church.

The family, the friends, the community in which he lived, his patients and the organizations to which he belonged will sorely miss this scholarly, friendly and talented man. Splendidly trained, possessed of a keen intellect and having the utmost regard for the nobility of his calling, it is not strange that he was respected throughout his State and section as an outstanding physician. Patients were drawn from great distance by his well deserved reputation.

A man of many social graces and broad intellectual interests, he touched the lives of many people. Those of us who knew him were benefited by the warmth and glow of his character. It is hard to believe that we will not again hear the cheery greeting and receive the warm handclasp of this dynamic, scholarly and sincere man. His patients loved him, his family loved him; and his friends loved him! His memory will ever remain graven on our hearts.

PAUL F. WHITAKER, M.D., F.A.C.P,
Member of Board of Governors for North Carolina

POSTGRADUATE COURSES, AUTUMN, 1944

THE AMERICAN COLLEGE OF PHYSICIANS

The following courses have been arranged through the generous cooperation of the directors and the institutions at which the courses will be given. The Advisory Committee on Postgraduate Courses will plan other courses during the winter and spring of 1945. Because of conditions due to the War, the program cannot be planned a long time in advance, but the College will attempt to provide an adequate program of postgraduate study to serve those members of the College who will find it convenient and possible to pursue refresher courses.

The courses are organized especially for Fellow and Associates of the College, but where facilities are available, they will be open to non-members with adequate preliminary training, preference to be given to non-members in the following order: (1) candidates for membership; (2) Medical Officers in the Armed Forces; (3) physicians preparing for examinations by their certifying boards; (4) other non-members having adequate background for advanced work. By direction of the Board of Regents, registrations from non-members of the College may not be accepted more than three weeks in advance of the opening of any course.

The courses are made available by the College to its members at minimum cost, because the College assumes full responsibility for promotion, advertising, printing and registration.

FEES—$20.00 per week to members of the College; $40.00 per week to non-members; Medical Officers of the Armed Forces of the United States and Canada, free. At least one-half of the registration fee shall be paid at time of filing application; the balance shall be paid not later than one week in advance of the opening of any course. Note that in the case of Course No. 3 a special, all-inclusive rate for tuition, room and board will apply: namely, $50.00 for members; $70.00 for non-members; $30.00 for Service physicians.

The advance payment may be refunded by the College to any registrant who for adequate reason is unable to pursue the course, providing notice of withdrawal is registered ten days in advance of the opening of the course.

REGISTRATION—All registrations should be filed directly with the Executive Offices of the American College of Physicians, 4200 Pine Street, Philadelphia 4, Pa. An official registration form, copy of which can be procured from the Executive Offices, must be used. Registrations will be assigned in order of receipt.

The College will record all registrations with the respective institutions offering the courses. Therefore, no registration should be made except through the American College of Physicians. A matriculation card will be sent to each registrant when his fee has been paid in full.

COURSE NO. 1—CARDIOLOGY

(October 2–7, 1944)

MASSACHUSETTS GENERAL HOSPITAL

Boston, Mass.

PAUL D. WHITE, M.D., F.A.C.P., *Director*

(Minimal Registration, 30; Maximal Registration, 50)

The full details of this course have already been published in the August issue of the ANNALS OF INTERNAL MEDICINE, and, therefore, are not hereunder repeated.

COURSE NO. 2—GENERAL MEDICINE

(October 9–14, 1944)

UNIVERSITY OF OREGON MEDICAL SCHOOL

Portland, Ore.

HOMER P. RUSH, M.D., F.A.C.P., *Director*

(Minimal Registration, 25; Maximal Registration, 60)

OFFICERS OF INSTRUCTION

Guests

Captain Ercell A. Addington, MC, Barnes Hospital.
Major Noyes L. Avery, Jr., MC, Barnes Hospital.
Colonel Charles K. Berle, MC, F.A.C.P., Barnes Hospital.
Major Frederick J. Bradshaw, Jr., MC, Barnes Hospital.
Commander L. T. Coggeshall, MC, USNR, Marine Bks., Klamath Falls.
Captain William W. Waddell, MC, Barnes Hospital.

David W. E. Baird, M.D., F.A.C.P., Dean, University of Oregon Medical School.
William Y. Burton, M.D., Assistant Professor of Radiology.
T. Homer Coffen, M.D., F.A.C.P., Clinical Professor of Medicine.
William S. Conklin, M.D., Assistant Professor of Medicine.
Norman A. David, M.D., Professor of Pharmacology.
Knox H. Finley, M.D., Associate Professor of Psychiatry.
John H. Fitzgibbon, M.D., F.A.C.P., Assistant Clinical Professor of Medicine.
Wesley E. Gatewood, M.D., Assistant Clinical Professor of Medicine.
Morton J. Goodman, M.D., Assistant Clinical Professor of Medicine.
John R. Hand, M.D., Assistant Clinical Professor of Urology.
Hance F. Haney, M.D., Professor of Physiology.
Blair Holcomb, M.D., F.A.C.P., Assistant Clinical Professor of Medicine.
Warren C. Hunter, M.D., Professor of Pathology.
Selma Hyman, M.D., Assistant Professor of Radiology.
Ralph C. Matson, M.D., F.A.C.P., Associate Clinical Professor of Medicine.
Sidney Mayer, Jr., M.D., Clinical Associate in Medicine.
John D. McGovern, M.D., Assistant Professor of Pathology.
Frank R. Menne, M.D., F.A.C.P., Professor of Pathology.
John R. Montague, M.D., Clinical Associate in Medicine.
Frank R. Mount, M.D., F.A.C.P., Assistant Clinical Professor of Medicine.
Edwin E. Osgood, M.D., F.A.C.P., Associate Professor of Medicine.
Dorwin L. Palmer, M.D., Assistant Clinical Professor of Radiology.
Matthew C. Riddle, M.D., Associate Professor of Medicine.
Homer P. Rush, M.D., F.A.C.P., Associate Clinical Professor of Medicine.
Laurence Selling, M.D., F.A.C.P., Professor of Medicine.
James T. Speros, M.D., Assistant Professor of Medicine.
Kenneth C. Swan, M.D., Assistant Professor of Ophthalmology.
Wilbur R. Todd, M.D., Assistant Professor of Biochemistry.
Ben Vidgoff, M.D., Clinical Instructor in Medicine.
Charles C. Wilson, M.D., Clinical Associate in Medicine.
Ivan M. Woolley, M.D., Clinical Associate in Radiology.

OUTLINE OF COURSE

Monday, October 9.

A.M. Session

8:00– 8:50 Registration, Introduction and Instructions.
Drs. Baird, Rush and Selling.

9:00– 9:50 X-Ray Interpretation of Diseases within the Thorax.
Drs. Palmer, Burton, Hyman and Woolley.

10:00–10:50 Chemotherapy: Penicillin.
Dr. Osgood.

11:00–11:50 Recent Advances in Cardiology.
Dr. Coffen.

P.M. Session

1:00– 2:50 Clinical Conference.
Dr. Selling.

3:00– 3:50 The Scope of Tropical Medicine: its subject content.
Dr. Riddle.

4:00– 4:50 The Psychic Factors in Asthma.
Dr. Mayer.

5:00– 5:50 Puberty Praecox and Associated Syndromes.
Dr. Hand.

Tuesday, October 10.

A.M. Session

8:00– 8:50 Clinical Pathological Conference.
Drs. Hunter, Menne and McGovern.

9:00– 9:50 X-Ray Interpretation of Diseases of the Gastrointestinal Tract.
Drs. Palmer, Burton, Hyman and Woolley.

10:00–10:50 Chemotherapy: The Sulphanilamides.
Dr. Osgood.

11:00–11:50 The Combination Use of Insulins.
Dr. Holcomb.

P.M. Session

1:00– 1:50 Functional Heart Disturbances: The Tension State.
Dr. Selling.

2:00– 2:50 Electroencephalograph.
Dr. Finley.

3:00– 3:50 The Scope of Tropical Medicine: The Biological and Sociological Factors.
Dr. Riddle.

4:00– 4:50 The Importance of Eye Ground Examination in Internal Medicine.
Dr. Goodman.

5:00– 5:50 The Eye and Diabetes.
Dr. Swan.

Wednesday, October 11.

A.M. Session

8:00– 8:50 Clinical Pathological Conference.
Drs. Hunter, Menne and McGovern.

9:00– 9:50 X-Ray Interpretation of Diseases of the Bones.
Drs. Palmer, Burton, Hyman and Woolley.

10:00–10:50 Chemotherapy: The Arsenicals.
Dr. Osgood.
11:00–11:50 The Value and Interpretation of Gastroscopic Examinations.
Dr. Fitzgibbon.

P.M. Session

1:00– 2:50 Clinical Conference.
Dr. Rush.
3:00– 3:50 Useful Diagnostic Procedures in Tropical Medicine.
Dr. Riddle.
4:00– 4:50 The Present Status of Estrogenic Therapy.
Dr. Vidgoff.
5:00– 5:50 Arthritis; a Workable Classification.
Dr. Wilson.

Thursday, October 12.

A.M. Session

8:00– 8:50 Clinical Pathological Conference.
Drs. Hunter, Menne and McGovern.
9:00– 9:50 X-Ray Interpretation of Diseases of the Genito-urinary Tract.
Drs. Burton, Palmer, Hyman and Woolley.
10:00–10:50 The Present Status of Thyroid Depressing Drugs.
Dr. David.
11:00–11:50 Clinical Experience with Thiouracil (Deracil Lederle).
Drs. Wilson and Montague.

P.M. Session will be spent at Barnes General Hospital. The Army program has been arranged by Colonel Charles K. Berle, MC, Commanding.

1:00– 1:45 Certain Problems when Tropical Diseases are moved to the Temperate Zone.
Commander Coggeshall.
1:45– 2:30 Physiology of Chest Leads.
Major Avery.
2:30– 3:15 Reconditioning, both Mental and Physical, as practiced at this Hospital, with Special Consideration of War Neuroses.
Major Bradshaw.
3:15– 4:00 Bronchogenic Carcinoma.
Captain Addington.
4:00– 4:45 Some Points in Diagnosis of Amoebic Hepatitis with Illustrative Cases.
Captain Waddell.

After these papers, an opportunity will be given the attending doctors to visit the Hospital.

Friday, October 13.

A.M. Session

8:00– 8:50 Clinical Pathological Conference.
Drs. Hunter, Menne and McGovern.
9:00– 9:50 Irradiation Therapy.
Drs. Palmer, Burton, Hyman and Woolley.
10:00–10:50 Present Status of the Physiological Background of the Vitamins.
Dr. Todd.
11:00–11:50 The Present Status of the Physiological Mechanism of Hypertension.
Dr. Haney.

P.M. Session

1:00– 2:50 Clinical Conference.
Dr. Gatewood.

3:00– 3:50 Experiences in War Medical Problems.
Lt. Col. Mount.

4:00– 4:50 The Present Status of Androgen Therapy.
Dr. Vidgoff.

5:00– 5:50 Arthritis; Plans of Therapy.
Dr. Wilson.

Saturday, October 14.

A.M. Session

8:00–12:00 Clinical Conference: Chest Diseases.
Drs. Matson, Conklin and Speros.

COURSE NO. 3—INTERNAL MEDICINE

(October 9–14, 1944)

CENTER FOR CONTINUATION STUDY,

UNIVERSITY OF MINNESOTA

Minneapolis, Minn.

JULIUS M. NOLTE, *Director*

Center for Continuation Study

WILLIAM A. O'BRIEN, M.D., *Director,*

Postgraduate Medical Education

CECIL J. WATSON, M.D., F.A.C.P., *Head,*

Department of Medicine

EDWARD H. RYNEARSON, M.D., F.A.C.P.

Associate Professor of Medicine,

Mayo Foundation, Rochester

(Minimal Registration, 30; Maximal Registration, 50)

OFFICERS OF INSTRUCTION

George N. Aagaard, Jr., M.D., Instructor in Medicine, Medical School.
John M. Adams, M.D., Assistant Professor of Pediatries, Medical School.
Wallace D. Armstrong, M.D., Professor of Physiological Chemistry, Medical School.
Elexious T. Bell, M.D., Professor of Pathology, Medical School.
Clarence Dennis, M.D., Associate Professor of Surgery, Medical School.
Charles A. Evans, M.D., Assistant Professor of Bacteriology, Medical School.
Gerald T. Evans, M.D., Associate Professor of Medicine, Medical School.
Edmund B. Flink, M.D., Associate Professor of Medicine, Medical School.
Malcolm M. Hargraves, M.D., Instructor in Medicine, Mayo Foundation, Rochester.
Wallace E. Herrell, M.D., F.A.C.P., Assistant Professor of Medicine, Mayo Foundation, Rochester.

Horton C. Hinshaw, M.D., F.A.C.P., Assistant Professor of Medicine, Mayo Foundation, Rochester.
Frederick W. Hoffbauer, M.D., Associate Professor of Medicine, Medical School.
Max H. Hoffman, M.D., Clinical Assistant Professor of Medicine, Medical School.
F. Raymond Keating, Jr., M.D., Instructor in Medicine, Mayo Foundation, Rochester.
Edwin J. Kepler, M.D., F.A.C.P., Associate Professor of Medicine, Mayo Foundation, Rochester.
Bernard G. Lannin, M.D., Clinical Instructor in Surgery, Medical School.
Thomas Lowry, M.D., Clinical Assistant Professor of Medicine, Medical School.
Irvine McQuarrie, M.D., Professor of Pediatrics, Medical School.
J. Arthur Myers, M.D., F.A.C.P., Professor of Preventive Medicine and Public Health, Medical School.
Julius M. Nolte, Director, Center for Continuation Study, University of Minnesota.
William A. O'Brien, M.D., Director, Postgraduate Medical Education, University of Minnesota.
E. R. Rickard, M.D., Director, Influenza Research Laboratory, Minnesota State Department of Health, Minneapolis.
Leo G. Rigler, M.D., Professor of Radiology, Medical School.
Edward H. Rynearson, M.D., F.A.C.P., Associate Professor of Medicine, Mayo Foundation, Rochester.
Thomas H. Seldon, M.D., Instructor in Anesthesia, Mayo Foundation, Rochester.
Morse J. Shapiro, M.D., Clinical Associate Professor of Medicine, Medical School.
Wesley W. Spink, M.D., F.A.C.P., Associate Professor of Medicine, Medical School.
Richard L. Varco, M.D., Assistant Professor of Surgery, Medical School.
Owen H. Wangensteen, M.D., F.A.C.S., Professor of Surgery, Medical School.
Cecil J. Watson, M.D., F.A.C.P., Professor of Medicine, Medical School.

The purpose of this course is to review certain phases of internal medicine. It will be presented by representatives from the faculty of the Medical School, Minneapolis, and the Mayo Foundation, Rochester, Minn. The program will consist of lectures, clinics, round tables, clinical dialogues, conferences, discussions, demonstrations and motion pictures. The group will live in the Center for Continuation Study Building, where lectures will be given. Clinics and Conferences will be held in the University of Minnesota Hospitals on the campus. The medical library is located just a short distance from the Center for Continuation Study. Arrangements have been made to house a maximum of fifty physicians. Twenty single rooms with detached bath and fifteen double rooms with adjoining bath have been made available. Occupants of double rooms may designate the physician who is to occupy the room with them, or they will be assigned a roommate pending final approval. All meals will be served in the dining room of the building. Members of the military service on active duty will be charged only for their room and meals. The advantage of living together during the week cannot be overemphasized, as it eliminates transportation difficulties and provides ample time for group discussion.

Outline of Course

Monday October 9.

7:30 Breakfast.
8:30 Orientation.
Center for Continuation Study Program.
Mr. Nolte.
Continuation Course in Internal Medicine.
Drs. O'Brien and Watson.
Laboratory Methods used in the Study of Blood Diseases.
Dr. Flink.

Interpretation of the Peripheral Blood Count.
Dr. Hargraves.
Fundamental Aspects of the Anemias.
Dr. Watson.

12:30 Luncheon.

2:00 Diagnosis and Treatment of the Anemias.
Dr. Watson.
Transfusions with Blood and Blood Plasma.
Dr. Seldon.
Transfusion Reactions.
Dr. Flink.

6:30 Dinner.

Tuesday, October 10.

7:30 Breakfast.

8:30 Characteristics of Virus Disease Agents.
Dr. Evans.
Influenza.
Dr. Rickard.
Virus Respiratory Infections in Infancy and Childhood.
Dr. Adams.
Virus Pneumonias.
Dr. Spink.

12:30 Luncheon.

2:00 Case Finding in Tuberculosis.
Dr. Myers.
Chemotherapy of Tuberculosis.
Dr. Hinshaw.
Clinical Radiologic Dialogue—Respiratory Infections.
Drs. Spink and Rigler.

6:30 Dinner.

Wednesday, October 11.

7:30 Breakfast.

8:30 Clinical Conference—Penicillin Therapy.
Drs. Spink and Herrell.
Clinical Pathologic Conference.
Drs. Watson, Bell and Associates.

12:30 Luncheon.

2:00 Changes in Blood Electrolytes in Disease.
Dr. Kepler.
Disease of the Adrenals.
Dr. McQuarrie.
Disturbances of the Pituitary Gland.
Dr. Rynearson.

6:30 Group Dinner—Faculty and Course Members.

Thursday, October 12.

7:30 Breakfast.

8:30 Calcium, Phosphorus and Phosphatase Studies.
Dr. Armstrong.
Hyperparathyroidism.
Dr. Keating.

The Male Sex Hormones.
Dr. Hoffman.
Thiouracil Therapy.
Dr. Evans.

12:30 Luncheon.

2:00 Fundamental Aspects of Jaundice.
Dr. Watson.
Liver Biopsy and Liver Function.
Dr. Hoffbauer.
Diagnosis and Treatment of Jaundice.
Dr. Watson.

6:30 Dinner.

Friday, October 13.

7:30 Breakfast.

8:30 Medical-Surgical Problems:
Treatment of Gastric and Duodenal Ulcer.
Dr. Lannin.
Treatment of Ulcerative Colitis.
Dr. Dennis.
Preparation of Surgical Patients.
Dr. Varco.
Treatment of Tumors of the Bowel.
Dr. Wangensteen.

12:30 Luncheon.

2:00 Differential Diagnosis of Coronary Occlusion and Myocardial Infarction.
Dr. Aagaard.
Treatment of Congenital Heart Disease.
Dr. Shapiro.
Clinical Radiologic Dialogue—Bronchial Obstruction.
Drs. Lowry and Rigler.

6:30 Dinner.

Saturday, October 14.

7:30 Breakfast.

8:30 General Medical Clinics: Exhibition of Patients showing Conditions Reviewed during Course.
Drs. Watson and Associates.

12:30 Luncheon.
Certificates of Attendance.

COURSE NO. 4—ALLERGY

(October 16–21, 1944)

ROOSEVELT HOSPITAL

New York, N. Y.

ROBERT A. COOKE, M.D., F.A.C.P., *Director*

(Minimal Registration, 25; Maximal Registration, 50)

OFFICERS OF INSTRUCTION

Robert A. Cooke, M.D., F.A.C.P., Attending Physician and Director, Department of Allergy, Roosevelt Hospital.

Horace S. Baldwin, M.D., Assistant Professor of Clinical Medicine, Cornell University Medical College; Assistant Attending Physician and Chief of Allergy Clinic, New York Hospital.

Aaron Brown, M.D., Assistant Clinical Professor of Medicine and Chief of Allergy Clinic, New York University College of Medicine; Assistant Visiting Physician, Bellevue Hospital.

Robert Chobot, M.D., F.A.C.P., Assistant Professor of Clinical Pediatrics, New York Post-Graduate Medical School and Hospital, Columbia University; Chief of Pediatric Allergy, New York Post-Graduate Medical School and Hospital; Assistant Chief, Allergy Clinic, Roosevelt Hospital.

Russell Clark Grove, M.D., Associate Surgeon, Otolaryngology, Roosevelt Hospital.

Joseph Harkavy, M.D., Associate in Medicine, Columbia University College of Physicians and Surgeons; Associate Physician and Chief of Allergy Clinic, Mt. Sinai Hospital; Associate Physician, Montefiore Hospital.

Selian Hebald, M.D., Assistant Chief of Allergy Clinic, Roosevelt Hospital; Senior Clinical Assistant in Allergy, Outpatient Department, Mt. Sinai Hospital.

Paul Klemperer, M.D., Pathologist, Mt. Sinai Hospital.

Louis Schwartz, M.D., Medical Director, Chief, Dermatoses Section, U. S. Public Health Service, Bureau of State Services, Bethesda, Md.

Will Cook Spain, M.D., F.A.C.P., Clinical Professor of Medicine, New York Post-Graduate Medical School and Hospital, Columbia University; Chief of Allergy Clinic and Attending Physician, New York Post-Graduate Medical School and Hospital.

Albert Vander Veer, M.D., Consultant in Allergy and Chief of Allergy Clinic, Roosevelt Hospital.

Matthew Walzer, M.D., Associate in Medicine, Cornell University Medical College; Attending in Allergy and Chief of Allergy Clinic, Jewish Hospital, Brooklyn.

The course in Allergy this year will be given in its entirety at the Roosevelt Hospital, but the faculty will be drawn from various medical schools and hospitals in New York and from the U. S. Public Health Service.

Instruction will be by means of lectures, clinics and conferences. All phases of allergy—immunological, pathological and clinical—will be considered from their practical and theoretical aspects. The course is planned to furnish the internist, general practitioner or allergist with the soundest and most recent information on concepts and procedures for diagnosis and treatment, and the general management of the allergic patient. On Saturday morning (October 21), there will be an optional morning session for those interested in preparation of allergenic extracts or desirous of practical experience in the technique of skin testing. A reference list of desirable articles on allergy will be mailed to all registrants from the office of the American College of Physicians.

Invitation to register for this particular course is extended beyond members of the American College of Physicians to members of the American Academy of Allergy, and to any members of the Inter-American Group who are pursuing postgraduate study in this Country at this time.

Outline of Course

Monday, October 16.

A.M. Session

9:00–11:30 Registration.
Introduction to Allergy.
Dr. Cooke.

11:30– 1:00 Extracts: Methods of Preparation and Standardization.
Dr. Spain.

P.M. Session

2:00– 4:00 Techniques of Skin Testing and Their Interpretation.
Dr. Walzer.

4:00– 6:00 Histopathology of the Allergic Reaction.
Dr. Klemperer.

7:30 Informal Dinner.

Tuesday, October 17.

A.M. Session

9:00–11:30 Pediatric Allergy (1st session).
Dr. Chobot.

11:30– 1:00 Allergic Coryza-Perennial.
Dr. Vander Veer.

P.M. Session

2:00– 4:00 Non-Infective Asthma.
Dr. Spain.

4:00– 6:00 Allergic Coryza-Seasonal (1st session).
Dr. Vander Veer.

Wednesday, October 18.

A.M. Session

9:00–10:30 Allergic Coryza-Seasonal (2nd session).
Dr. Brown.

10:30–11:30 Allergic Coryza-Seasonal, Special Features.
Dr. Hebald.

11:30– 1:00 Vascular Allergy; Meniere's Disease; Migraine; Physical Allergy.
Dr. Harkavy.

P.M. Session

2:00– 4:00 Industrial Dermatoses.
Dr. Schwartz.

4:00– 6:00 Sinus Disease in Relation to Allergy.
Dr. Grove.

Thursday, October 19.

A.M. Session

9:00–10:30 Serum Disease; Drug and Insulin Allergy.
Dr. Harkavy.

10:30–12:30 Asthma: Differential Diagnosis; Symptomatic Treatment of Status Asthmaticus.
Dr. Baldwin.

P.M. Session

2:00– 3:30 Pediatric Allergy (2nd session).
Dr. Chobot.

3:30– 6:00 Infective Asthma.
Dr. Cooke.

Friday, October 20.

A.M. Session

9:00–12:30 Eczema, Urticaria, Angioneurotic Edema and Miscellaneous Allergies, with Case Presentations.
Dr. Cooke.

P.M. Session

2:00– 5:00 General Review and Roundtable.

By coincidence, the American College of Physicians will be conducting a Regional Meeting for the State of New York in New York City, Friday, October 20. Announcements concerning the program will be furnished to all registrants in this course. However, all are invited to attend the regional dinner meeting from 6:00 to 8:00 P.M. at the Waldorf-Astoria Hotel, and to be the guests of the New York Academy of Medicine at 8:30 P.M. at a Panel Discussion on "The Evaluation of Sulfa Drugs and Penicillin," a feature of the Seventeenth Graduate Fortnight at the Academy. Dr. David P. Barr, F.A.C.P., will be the Chairman of the Panel Discussion, and Drs. Rene J. Dubos, Colin M. MacLeod, John F. Mahoney, Frank L. Meleney and William S. Tillett will assist.

Saturday, October 21.

A.M. Session

9:00 Optional. Practical Work, Tests, Extracts and Clinic Management.
Drs. Cooke and Spain.

COURSE NO. 5—SPECIAL PHASES OF INTERNAL MEDICINE

(October 23–November 4, 1944)

CHICAGO INSTITUTIONS

THORNE HALL

NORTHWESTERN UNIVERSITY

Lake Shore Drive and Superior Street

Chicago, Ill.

WILLARD O. THOMPSON, M.D., F.A.C.P., *Director*

WALTER L. PALMER, M.D., F.A.C.P., *Governor for Northern Illinois*

(Minimal Registration, 25; Maximal Registration, 60)

This course offers an unusual opportunity for physicians to familiarize themselves with recent developments in various fields of Internal Medicine. The faculty of 110 men includes many of the most outstanding physicians, not only in Chicago, but also from other parts of the country. Numerous Chicago Institutions are represented, including the University of Chicago, University of Illinois, Northwestern University,

Loyola University, the Institute of Psychoanalysis, the Illinois Neuropsychiatric Institute, the Michael Reese Hospital, as are also many outside agencies and institutions, including Harvard Medical School, Ohio State University, University of Michigan, University of Minnesota, Washington University, University of Oklahoma, University of Cincinnati, State University of Iowa, Marquette University, the Cleveland Clinic, Indianapolis City Hospital, the Medical Corps of the U. S. Army and the U. S. Navy, and the U. S. Public Health Service. Where possible, clinical discussions will be illustrated by demonstration of patients.

Meeting Place: Thorne Hall, Northwestern University, Lake Shore Drive and Superior Street—All sessions will be held in Thorne Hall, except those on Saturday, November 4, which will be held in the Drake Hotel. This day will be devoted to the Regional Meeting of the American College of Physicians for the States of Illinois, Indiana, Iowa, Kentucky, Michigan, Minnesota and Wisconsin. This meeting is part of the course, and all physicians taking the course, whether they be members of the College or not, are invited not only to participate in the scientific sessions of the Regional Meeting, but also to attend the luncheon and the dinner.

Luncheon: Abbott Hall, Northwestern University, 710 Lake Shore Drive—Abbott Hall is just across the street from Thorne Hall.

Officers of Instruction

William E. Adams, M.D., F.A.C.S., Associate Professor of Surgery, University of Chicago, The School of Medicine.

Wright R. Adams, M.D., Associate Professor of Medicine, University of Chicago, The School of Medicine.

Edgar V. Allen, F.A.C.P., Col., (MC), AUS, Consultant in Medicine, Seventh Service Command, Omaha, Nebr.

Raymond B. Allen, M.D., Dean, University of Illinois College of Medicine.

Howard Alt, M.D., Assistant Professor of Medicine, Northwestern University Medical School.

Edwin B. Astwood, M.D., Assistant Professor of Pharmacotherapy, Harvard Medical School, Boston, Mass.

Percival Bailey, M.D., Professor of Neurology and Neurosurgery, University of Illinois College of Medicine.

Clifford Barborka, M.D., F.A.C.P., Assistant Professor of Medicine and Director, Abbott Foundation for Medical Research, Northwestern University Medical School.

Claude S. Beck, F.A.C.S., Lt. Col., (MC), AUS, Consultant in Surgery, Fifth Service Command, Columbus, Ohio.

Granville Bennett, M.D., Professor of Pathology, University of Illinois College of Medicine.

Robert S. Berghoff, M.D., F.A.C.P., Clinical Professor of Medicine, Loyola University School of Medicine; President-Elect, Illinois State Medical Society.

Bert I. Beverly, M.D., Assistant Professor of Pediatric Psychiatry, University of Illinois College of Medicine.

Marion A. Blankenhorn, M.D., F.A.C.P., Professor of Medicine and Head of the Department, University of Cincinnati College of Medicine, Cincinnati, Ohio.

William J. Bleckwenn, Col., (MC), AUS, Consultant in Neuropsychiatry, Sixth Service Command, Chicago. (Formerly, Surgeon in the forward echelon, Southwest Pacific Area.)

Robert G. Bloch, M.D., F.A.C.P., Professor of Medicine and Chief of the Division of Pulmonary Diseases, University of Chicago, The School of Medicine.

Alexander Brunschwig, M.D., F.A.C.S., Professor of Surgery, University of Chicago, The School of Medicine.

Paul R. Cannon, M.D., Professor and Chairman of the Department of Pathology, University of Chicago, The School of Medicine.

Charles M. Caravati, F.A.C.P., Lt. Col., (MC), AUS, Physician-in-Chief, Percy Jones General Hospital, Battle Creek, Mich.

Anton J. Carlson, M.D., F.A.C.P., Professor of Physiology, Emeritus, University of Chicago, The School of Medicine.

Warren H. Cole, M.D., F.A.C.S., Professor and Head of the Department of Surgery, University of Illinois College of Medicine.

Arthur R. Colwell, M.D., F.A.C.P., Assistant Professor of Medicine, Northwestern University Medical School.

Edward L. Compere, M.D., F.A.C.S., Associate Professor of Surgery, Northwestern University Medical School; Chairman, Department of Orthopedic Surgery, Wesley Memorial Hospital.

Robert M. Craig, M.D., Passed Assistant Surgeon (R), U. S. Public Health Service, Chicago.

Irving S. Cutter, M.D., Professor of Medicine, Emeritus, Northwestern University Medical School.

Israel Davidsohn, M.D., F.A.C.P., Assistant Professor of Pathology, University of Illinois College of Medicine; Pathologist and Director of Laboratories, Mt. Sinai Hospital.

Geza de Takats, M.D., F.A.C.S., Associate Professor of Surgery, University of Illinois College of Medicine.

Charles A. Doan, M.D., F.A.C.P., Professor of Medicine and Chairman of the Department, Ohio State University College of Medicine, Columbus, Ohio.

Lester R. Dragstedt, M.D., F.A.C.P., Professor of Surgery, University of Chicago, The School of Medicine.

Samuel M. Feinberg, M.D., F.A.C.P., Associate Professor of Medicine, Northwestern University Medical School.

Morris Fishbein, M.D., Assistant Professor of Medicine, University of Illinois College of Medicine; Editor, Journal of the American Medical Association.

Thomas M. Francis, M.D., Professor of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, Mich.

Thomas M. French, M.D., Assistant Director, Institute of Psychoanalysis.

Lee C. Gatewood, M.D., F.A.C.P., Professor of Medicine (Rush), University of Illinois College of Medicine.

Francis J. Gerty, M.D., Professor of Psychiatry and Head of the Department, University of Illinois College of Medicine; Director, Psychiatric Division, Illinois Neuropsychiatric Institute.

Stanley Gibson, M.D., Professor of Pediatrics and Head of the Department, Northwestern University Medical School.

Newell C. Gilbert, M.D., Professor of Medicine and Head of the Department, Northwestern University Medical School.

Carl G. Hartman, Ph.D., Professor and Head of the Department of Zoology and Physiology, University of Illinois, Urbana, Ill.

B. C. H. Harvey, M.D., Dean of Medical Students, The University of Chicago.

Jerome R. Head, M.D., Assistant Professor of Surgery, Northwestern University Medical School; Medical Director, Edward Sanitarium, Naperville, Ill.

Norris J. Heckel, M.D., F.A.C.S., Associate Professor of Urology (Rush), University of Illinois College of Medicine.

Don G. Hildrup, Colonel, (MC), USA, Surgeon, Sixth Service Command, Chicago, Ill.

Paul H. Holinger, M.D., F.A.C.S., Assistant Professor of Otolaryngology, University of Illinois College of Medicine.

Archibald L. Hoyne, M.D., F.A.C.P., Professor of Pediatrics, University of Illinois College of Medicine; Clinical Professor of Pediatrics, University of Chicago, The School of Medicine.

Charles B. Huggins, M.D., Professor of Surgery (Genito-urinary), University of Chicago, The School of Medicine.

Ernest E. Irons, M.D., F.A.C.P., Professor of Medicine, University of Illinois College of Medicine; President, American College of Physicians.

Raphael Isaacs, M.D., F.A.C.P., Attending Physician in Hematology and Director, Laboratory of Hematology, Michael Reese Hospital.

Andrew C. Ivy, M.D., F.A.C.P., Nathan Smith Davis Professor of Physiology, Northwestern University Medical School.

Louis N. Katz, M.D., F.A.C.P., Professorial Lecturer in Physiology, University of Chicago, The School of Medicine; Head of the Division of Cardiovascular Research, Michael Reese Hospital.

Robert Wood Keeton, M.D., F.A.C.P., Professor of Medicine and Head of the Department, University of Illinois College of Medicine.

Norman M. Keith, M.D., Professor of Medicine, Mayo Foundation, University of Minnesota; Consulting Physician, Mayo Clinic; Rochester, Minn.

Frank B. Kelly, M.D., F.A.C.P., Professor of Medicine (Rush), University of Illinois College of Medicine.

Edward C. Kendall, M.D., Professor of Biochemistry, Mayo Foundation, University of Minnesota, Rochester, Minn.

Allen T. Kenyon, M.D., Associate Professor of Medicine, University of Chicago, The School of Medicine.

Fred C. Koch, Ph.D., Professor of Biochemistry, Emeritus, University of Chicago.

Peter C. Kronfeld, M.D., Associate Professor of Ophthalmology and Director of Education, Illinois Eye & Ear Infirmary, University of Illinois College of Medicine.

Grant H. Laing, M.D., F.A.C.P., Assistant Professor of Medicine, Northwestern University Medical School.

Sidney O. Levinson, M.D., Director, Samuel Deutsch Serum Center, Michael Reese Hospital.

Louis R. Limarzi, M.D., Assistant Professor of Medicine, University of Illinois College of Medicine.

John R. Lindsay, M.D., Professor of Otolaryngology, University of Chicago, The School of Medicine.

Malcolm T. MacEachern, M.D., F.A.C.P., Associate Professor of Medicine, Northwestern University Medical School; Director of Program of Hospital Administration, Northwestern University School of Commerce; Chairman of the Administrative Board, American College of Surgeons.

Paul B. Magnuson, M.D., F.A.C.S., Professor of Bone and Joint Surgery and Head of the Department, Northwestern University Medical School.

Chauncey C. Maher, M.D., F.A.C.P., Associate Professor of Medicine, Northwestern University Medical School.

David E. Markson, M.D., F.A.C.P., Associate Professor of Medicine and Director of the Arthritis Clinic, Northwestern University Medical School.

Donald McCarthy, F.A.C.P., Captain, (MC), USN, Professional Executive Officer, U. S. Naval Hospital, Great Lakes, Ill.

E. P. McCullagh, M.D., F.A.C.P., Head of Endocrine Research Laboratory, Cleveland Clinic Foundation Hospital, Cleveland, Ohio.

Warren S. McCulloch, M.D., Associate Professor of Psychiatry, University of Illinois College of Medicine.

Ross T. McIntire, F.A.C.P., Vice Admiral, (MC), USN, Surgeon General, U. S. Navy, Washington, D. C.

Phillip Miller, M.D., Professor of Medicine, University of Chicago, The School of Medicine.

James H. Mitchell, M.D., Professor of Dermatology (Rush), University of Illinois College of Medicine.

Herman J. Moersch, M.D., F.A.C.P., Associate Professor of Medicine, Mayo Foundation, University of Minnesota, Rochester, Minn.

Carl R. Moore, Ph.D., Professor and Head of the Department of Zoology, University of Chicago.

Carl V. Moore, Jr., M.D., F.A.C.P., Associate Professor of Medicine, Washington University School of Medicine, St. Louis, Mo.

Josiah J. Moore, M.D., F.A.C.P., Director, Moore Clinical Laboratory; President, Chicago Medical Society.

Walter H. Nadler, M.D., Associate Professor of Medicine, Northwestern University Medical School.

Warren O. Nelson, M.D., Professor of Anatomy, State University of Iowa College of Medicine, Iowa City, Iowa.

Eric Oldberg, M.D., F.A.C.S., Professor of Neurology and Neurological Surgery and Head of the Department, University of Illinois College of Medicine.

Irvine H. Page, M.D., F.A.C.P., Director, Lilly Laboratory for Clinical Research, Indianapolis City Hospital, Indianapolis, Ind.

Walter L. Palmer, M.D., F.A.C.P., Professor of Medicine, University of Chicago, The School of Medicine.

Max M. Peet, M.D., F.A.C.S., Professor of Surgery, University of Michigan Medical School, Ann Arbor, Mich.

Carl M. Peterson, M.D., Secretary, Council on Industrial Health, American Medical Association.

Dallas B. Phemister, M.D., F.A.C.S., Thomas D. Jones Professor of Surgery and Head of the Department, University of Chicago, The School of Medicine.

Sidney A. Portis, M.D., F.A.C.P., Associate Professor of Medicine (Rush), University of Illinois College of Medicine.

Frank B. Queen, F.A.C.P., Lt. Col., (MC), AUS, Chief of Laboratory Service, Bushnell General Hospital, Brigham City, Utah.

Armand J. Quick, Ph.D., M.D., Professor of Biochemistry and Head of the Department, Marquette University School of Medicine, Milwaukee, Wis.

Paul S. Rhoads, M.D., F.A.C.P., Associate Professor of Medicine, Northwestern University Medical School.

Henry T. Ricketts, M.D., Associate Professor of Medicine, University of Chicago, The School of Medicine.

Stephen Rothman, M.D., Associate Professor of Medicine and Head of the Section of Dermatology and Syphilology, University of Chicago, The School of Medicine.

George J. Rukstinat, M.D., Professor of Pathology, University of Illinois College of Medicine.

Edward H. Rynearson, M.D., F.A.C.P., Associate Professor of Medicine, Mayo Foundation, University of Minnesota, Rochester, Minn.

George X. Schwemlein, M.D., Passed Assistant Surgeon (R), U. S. Public Health Service, Chicago.

George W. Scupham, M.D., Associate Professor of Medicine, Northwestern University Medical School.

Francis E. Senear, M.D., F.A.C.P., Professor of Dermatology and Head of the Department, University of Illinois College of Medicine.

James P. Simonds, M.D., Professor of Pathology, Emeritus, Northwestern University Medical School.

David Slight, M.D., Professor of Psychiatry, University of Chicago, The School of Medicine.

LeRoy H. Sloan, M.D., F.A.C.P., Professor of Medicine, University of Illinois College of Medicine.

Lowell D. Snorf, M.D., F.A.C.P., Associate Professor of Medicine, Northwestern University Medical School.

Samuel Soskin, M.D., Professorial Lecturer in Physiology, University of Chicago, The School of Medicine; Medical Director, Michael Reese Hospital.

Ralph Soto-Hall, Major, (MC), AUS, Consultant in Orthopedic Surgery, Sixth Service Command, Chicago. (Formerly, Consultant in the European Theater of Operations.)

Wesley W. Spink, M.D., F.A.C.P., Associate Professor of Medicine, University of Minnesota Medical School, Minneapolis, Minn.

Henry C. Sweany, M.D., F.A.C.P., Associate Professor of Medicine, Northwestern University Medical School; Professorial Lecturer in the Department of Pharmacology, University of Chicago; Medical Director of Research and Laboratories, Municipal Tuberculosis Sanitarium.

Frederic E. Templeton, M.D., Cleveland Clinic, Cleveland, Ohio.

Willard O. Thompson, M.D., F.A.C.P., Professor of Medicine (Rush), University of Illinois College of Medicine.

Henry H. Turner, M.D., F.A.C.P., Associate Professor of Medicine, University of Oklahoma School of Medicine, Oklahoma City, Okla.

Theodore R. Van Dellen, M.D., F.A.C.P., Associate Professor of Medicine and Head of the Cardiac Clinic, Northwestern University Medical School.

Adrien H. P. E. Verbrugghen, M.D., F.A.C.S., Associate Professor of Neurological Surgery (Rush), University of Illinois College of Medicine.

Italo F. Volini, M.D., F.A.C.P., Professor and Head of the Department of Medicine and Dean of the School of Medicine, Loyola University.

George E. Wakerlin, M.D., F.A.C.P., Professor of Physiology and Head of the Department, University of Illinois College of Medicine.

Cecil J. Watson, M.D., F.A.C.P., Professor of Medicine and Head of the Department, University of Minnesota Medical School, Minneapolis, Minn.

Irving S. Wright, F.A.C.P., Lt. Col., (MC), AUS, Consultant in Medicine, Sixth Service Command, Chicago.

Frederick A. Willius, M.D., F.A.C.P., Head of the Section on Cardiology, Mayo Clinic; Associate Professor of Medicine, Mayo Foundation, University of Minnesota; Rochester, Minn.

Outline of Course

Monday, October 23.

Cardiovascular and Renal Diseases

Willard O. Thompson, M.D., F.A.C.P., *Presiding*

A.M. Session

7:30– 8:30 Registration.
Thorne Hall, Lake Shore Drive and Superior Street.

8:30– 9:00 Diagnosis of Congenital Heart Disease.
Dr. W. R. Adams.

9:00– 9:30 Rheumatic Fever in Children.
Dr. Gibson.

9:30–10:00 Rheumatic Fever: Correlation of Clinical and Pathologic Findings.
Dr. Simonds.

10:00–10:30 Rheumatic Heart Disease. The Sequence of Pathologic Changes as They Affect the Heart.
Dr. Volini.

10:30–10:45 Intermission—Refreshments.
10:45–11:15 Rheumatic Fever in the Army.
Lt. Col. Wright.
11:15–11:45 Rheumatic Fever in the Navy.
Capt. McCarthy.
11:45–12:15 Rheumatic Carditis.
Dr. Willius.
12:15–12:45 Management of Cardiac Edema.
Dr. Maher.
12:45– 1:15 Pathogenesis of Subacute Bacterial Endocarditis.
Dr. Willius.
1:15– 2:15 Luncheon.

Monday October 23.

Cardiovascular and Renal Diseases (Continued)

ITALO F. VOLINI, M.D., F.A.C.P., *Presiding*

P.M. Session
2:15– 2:45 The Factors Influencing Recovery or Death in Acute Coronary Occlusion.
Dr. Willius.
2:45– 3:15 Clinical Aspects of the Control of Coronary Circulation.
Dr. Katz.
3:15– 3:45 The Surgical Approach to Coronary Disease.
Lt. Col. Beck.
3:45– 4:15 Constrictive Pericarditis.
Dr. Willius.
4:15– 4:30 Intermission—Refreshments.
4:30– 5:00 Compression of the Heart.
Lt. Col. Beck.
5:00– 5:30 Less Familiar Vascular Syndromes.
Dr. Sloan.
5:30– 5:50 Discussion.

Tuesday, October 24.

Cardiovascular and Renal Diseases (Continued)

MORRIS FISHBEIN, M.D., *Presiding*

A.M. Session
8:00– 8:30 Recent Advances in the Utility of the Electrocardiogram in Clinical Practice.
Dr. Katz.
8:30– 9:00 Surgical Treatment of Hypertension.
Dr. Peet.
9:00– 9:30 Essential Hypertension: Clinical Groups and Their Course.
Dr. Keith.
9:30–10:00 Hypertension and Bright's Disease.
Dr. Page.
10:00–10:30 Hypertension Heart Disease.
Dr. Nadler.
10:30–10:45 Intermission—Refreshments.
10:45–11:15 Arteriosclerosis.
Dr. Van Dellen.

11:15–11:45 Acute Nephritis Including Acute Renal Insufficiency; Course and Prognosis.
Dr. Keith.
11:45–12:15 Diagnosis and Treatment of Common Urinary Infections.
Dr. Heckel.
12:15–12:45 Chronic Glomerulonephritis; Course and Prognosis.
Dr. Keith.
12:45– 1:15 Ophthalmoscopy in the Diagnosis of Human Illness.
Dr. Kronfeld.
1:15– 2:15 Luncheon.

Tuesday, October 24.

Cardiovascular and Renal Diseases (Continued)

DON G. HILDRUP, Colonel, (MC), USA, *Presiding*

P.M. Session
2:15– 2:45 Newer Concepts Regarding Uremia.
Dr. Keith.
2:45– 3:25 Peripheral Vascular Disease.
Lt. Col. Wright.
3:25– 3:55 Surgical Treatment of Peripheral Vascular Disease.
Dr. de Takats.
3:55– 4:15 Sympathectomy in the Treatment of Peripheral Vascular Disease.
Dr. Peet.
4:15– 4:30 Intermission—Refreshments.
4:30– 5:00 Periarteritis Nodosa.
Dr. Scupham.
5:00– 5:30 Shock.
Dr. Page.
5:30– 5:50 Discussion.

Wednesday, October 25.

Arthritis

JOSIAH J. MOORE, M.D., F.A.C.P., *Presiding*

A.M. Session
8:00– 8:30 Surgical Phases of Arthritis.
Dr. Compere.
8:30– 9:00 Classification and Treatment of Arthritis.
Dr. Markson.
9:00– 9:30 Comparison of Different Forms of Arthritis.
Dr. Bennett.
9:30–10:00 The Differential Diagnosis of Conditions that Cause Low Back Pain Accompanied by Sciatica.
Dr. Magnuson.

Industrial Medicine

10:00–10:30 Industry Needs the Physician.
Dr. Peterson.
10:30–10:45 Intermission—Refreshments.

Pulmonary Diseases

10:45–11:15 Tuberculosis Control in General Medical Practice.
Dr. Bloch.

11:15–11:45 Surgical Treatment of Pulmonary Tuberculosis.
Dr. Head.
11:45–12:15 Diagnosis of Non-Tuberculous Pulmonary Lesions with Special Reference to Silicosis.
Dr. Sweany.
12:15–12:45 Aspiration Pneumonia.
Dr. Irons.
12:45– 1:15 The Confusing Clinical Picture of Atypical Primary Bronchial Pneumonia.
Dr. Rhoads.
1:15– 2:15 Luncheon.

Wednesday, October 25.

Pulmonary Diseases (Continued)

LEROY H. SLOAN, M.D., F.A.C.P., *Presiding*

P.M. Session
2:15– 2:45 Differential Diagnosis and Treatment of Intrathoracic Tumors.
Dr. W. E. Adams.
2:45– 3:30 Bronchial Tumors.
Dr. Holinger.

Neurology and Psychiatry

3:30– 4:00 Pathology and Symptomatology of the Hypothalamus.
Dr. Bailey.
4:00– 4:15 Intermission—Refreshments.
4:15– 4:45 Intervertebral Discs.
Dr. Oldberg.
4:45– 5:15 Meniere's Syndrome.
Dr. Lindsay.
5:15– 5:45 Relation of Psychoanalysis to Internal Medicine.
Dr. French.

Thursday, October 26.

Neurology and Psychiatry (Continued)

WALTER L. PALMER, M.D., F.A.C.P., *Presiding*

A.M. Session
8:00– 8:30 Some Physiological Aspects of Mental Disorder.
Dr. McCulloch.
8:30– 9:00 Psychological Problems in the General Hospital.
Dr. Gerty.
9:00– 9:30 Psychological Problems of the Adolescent.
Dr. Beverly.
9:30–10:00 Psychotherapy in General Medicine.
Dr. Slight.

Gastrointestinal Diseases

ERNEST E. IRONS, M.D., F.A.C.P., *Presiding*

10:00–10:30 Management of Peptic Ulcer.
Dr. Palmer.
10:30–10:45 Intermission—Refreshments.

10:45–11:30 Management of Peptic Ulcer (Continued).
Dr. Palmer.
11:30–12:00 The Technics of Roentgenologic Examination of the Oesophagus, Stomach and Duodenum.
Dr. Templeton.
12:00–12:45 Recent Contributions to the Physiology of the Gastrointestinal Tract.
Dr. Ivy.
12:45– 1:15 The Normal Oesophagus, Stomach and Duodenum.
Dr. Templeton.
1:15– 2:15 Luncheon.

Thursday, October 26.

Gastrointestinal Diseases (Continued)

WALTER L. PALMER, M.D., F.A.C.P., *Presiding*

P.M. Session
2:15– 2:45 Diagnosis and Treatment of Oesophageal Disease.
Dr. Moersch.
2:45– 3:15 Surgical Treatment of Lesions of Lower Oesophagus.
Dr. Phemister.
3:15– 3:45 Abnormalities of the Oesophagus, Stomach and Duodenum.
Dr. Templeton.
3:45– 4:15 Differential Diagnosis of Cardiospasm.
Dr. Moersch.
4:15– 4:30 Intermission—Refreshments.
4:30– 5:00 Inflammatory Conditions of the Oesophagus, Stomach and Duodenum.
Dr. Templeton.
5:00– 5:45 Clinico-Pathologic Conference.
Drs. Sloan and Simonds.

Friday, October 27.

Gastrointestinal Diseases (Continued)

GRANT H. LAING, M.D., F.A.C.P., *Presiding*

A.M. Session
8:00– 8:30 Relation between Deficiency Diseases and the Gastrointestinal Tract.
Dr. Barborka.
8:30– 9:30 Natural History of Carcinoma of the Stomach.
Dr. Palmer.
9:30–10:00 The Value of Gastroscopy in the Study of Gastric Distress following Gastric Surgery.
Dr. Moersch.
10:00–10:30 Dyspepsia.
Dr. Palmer.
10:30–10:45 Intermission—Refreshments.

WALTER L. PALMER, M.D., F.A.C.P., *Presiding*

10:45–11:15 Surgical Treatment of Peptic Ulcer.
Dr. Cole.
11:15–11:45 Section of the Vagus Nerve in the Treatment of Gastro-duodenal Ulcer.
Dr. Dragstedt.
11:45–12:15 Isolated Ulcerative Lesions of the Intestine.
Lt. Col. Caravati.

12:15–12:45 Non-specific Ulcerative Colitis.
Dr. Snorf.
12:45– 1:15 The Extension of Surgical Attack upon Advanced Intro-abdominal Cancer.
Dr. Brunschwig.
1:15– 2:15 Luncheon.

Friday, October 27.

Gastrointestinal Diseases (Continued)

WALTER L. PALMER, M.D., F.A.C.P., *Presiding*

P.M. Session
2:15– 2:45 Amebiasis.
Dr. Gatewood.
2:45– 3:15 Psychosomatic Aspects of the Gastrointestinal Tract.
Dr. Portis.
3:15– 3:45 Surgical Treatment of Ulcerative Colitis.
Dr. Dragstedt.
3:45– 4:15 Diseases of the Gall Bladder.
Dr. Cole.
4:15– 4:30 Intermission—Refreshments.
4:30– 5:15 Applied Physiology of the Gall Bladder.
Dr. Ivy.
5:15– 5:45 Hepatitis.
Lt. Col. Caravati.

Saturday, October 28.

Gastrointestinal Diseases (Continued)

PAUL S. RHOADS, M.D., F.A.C.P., *Presiding*

A.M. Session
8:00– 8:30 Cirrhosis of the Liver.
Dr. Nadler.
8:30– 9:15 Liver Function Tests.
Dr. Ivy.
9:15–10:00 The Protein Problem.
Dr. Cannon.

Dermatology

10:00–10:30 Dermatitis Medicamentosa.
Dr. Senear.
10:30–10:45 Intermission—Refreshments.
10:45–11:15 Cutaneous Diseases in Relation to Internal Disturbances.
Dr. Rothman.
11:15–11:45 Fungus Infections of the Skin.
Dr. Mitchell.
11:45–12:15 Modern Conceptions of Eczema.
Dr. Senear.
12:15–12:45 Rôle of Allergy in Dermatology.
Dr. Feinberg.
12:45– 1:15 Recent Advances in the Treatment of Syphilis.
Drs. Craig and Schwemlein.
1:15– 2:15 Luncheon.

Monday, October 30.

Endocrinology

WILLARD O. THOMPSON, M.D., F.A.C.P., *Presiding*

A.M. Session

8:00– 8:30 Selection of Insulin in Diabetic Therapy.
Dr. Colwell.
8:30– 9:00 Treatment of Diabetic Acidosis.
Dr. Ricketts.
9:00– 9:30 The Surgery of Malignant Pancreatic Tumors.
Dr. Brunschwig.
9:30–10:30 Practical Considerations in the Management of Diabetic Patients (Clinic).
Dr. Keeton.
10:30–10:45 Intermission—Refreshments.
10:45–11:45 Practical Considerations in the Management of Diabetic Patients (Clinic Continued).
Dr. Keeton.
11:45–12:15 Types of Diabetes Mellitus and their Treatment.
Dr. Colwell.
12:15–12:45 Hyperinsulinism.
Dr. Rynearson.
12:45– 1:15 Production of Pseudohermaphroditism.
Dr. Ivy.
1:15– 2:15 Luncheon.

Monday, October 30.

Endocrinology (Continued)

MALCOLM T. MACEACHERN, M.D., F.A.C.P., *Presiding*

P.M. Session

2:15– 3:15 The Normal and Hyperactive Ovary in Relation to the Menstrual Cycle.
Dr. Hartman.
3:15– 3:45 Carcinoma of the Prostate.
Dr. Huggins.
3:45– 4:15 Evaluation of Sex Hormones in the Treatment of Some Urologic Diseases.
Dr. Heckel.
4:15–4:30 Intermission—Refreshments.
4:30– 5:30 Diseases of the Pituitary.
Dr. Rynearson.
5:30– 5:50 Discussion.

Tuesday, October 31.

Endocrinology (Continued)

IRVING S. CUTTER, M.D., *Presiding*

A.M. Session

8:00– 8:30 Factors in Puberty.
Dr. Kenyon.
8:30– 9:00 Endocrine Factors in Human Puberty.
Dr. Nelson.

9:00–10:00 The Interpretation of Data on the Male Sex Hormone; 17-Keto-Steroid Content of Human Urine.
Dr. Koch.
10:00–10:30 Addison's Disease, Emphasizing Diagnostic Tests and Limitations of Desoxycorticosterone Therapy.
Dr. McCullagh.
10:30–10:45 Intermission—Refreshments.
10:45–11:15 Surgical Diagnosis of Pituitary Tumor.
Dr. Verbrugghen.
11:15–11:45 Some Clinical Aspects of Dwarfism.
Dr. McCullagh.
11:45–12:30 Endocrine Clinic: Hypogonadism.
Dr. Thompson.
12:30– 1:15 Endocrine Organs in Early Life and Some Associated Disorders.
Dr. C. R. Moore.
1:15– 2:15 Luncheon.

Tuesday, October 31.

Endocrinology (Continued)

RAYMOND B. ALLEN, M.D., *Presiding*

P.M. Session

2:15– 3:15 Endocrine Clinic: Hypogonadism (Continued).
Dr. Thompson.
3:15– 3:45 Disorders of Reproduction in the Human Male.
Dr. Nelson.
3:45– 4:15 Types of Testicular Failure and Their Management.
Dr. McCullagh.
4:15– 4:30 Intermission—Refreshments.
4:30– 5:00 Results of Seven Years of Clinical Experience with Testosterone Propionate.
Dr. Turner.
5:00– 5:30 Hormonal Therapy in Menstrual Disorders.
Dr. McCullagh.
5:30– 5:50 Discussion.

Wednesday, November 1.

Endocrinology (Continued)

GEORGE E. WAKERLIN, M.D., F.A.C.P., *Presiding*

A.M. Session

8:00– 9:00 Endocrine Clinic: Hypogonadism (Continued).
Dr. Thompson.
9:00– 9:30 Endocrine Physiology of the Breast.
Dr. Nelson.
9:30–10:00 Endocrine Dwarfism; Its Diagnosis and Treatment.
Dr. Turner.
10:00–10:30 Treatment of Dwarfism.
Dr. Thompson.
10:30–10:45 Intermission—Refreshments.
10:45–11:15 Treatment of Dwarfism (Continued).
Dr. Thompson.
11:15–11:45 Influence of the Adrenals on Carbohydrate Metabolism.
Dr. Kendall.

11:45–12:15 Experimental Production of Mammary Carcinoma.
Dr. Nelson.
12:15–12:45 Persistence of Estrogenic Effects after Discontinuance of Treatment.
Dr. Turner.
12:45– 1:15 Obesity.
Dr. Soskin.
1:15– 2:15 Luncheon.

Wednesday, November 1.

Endocrinology (Continued)

ROBERT W. KEETON, M.D., F.A.C.P., *Presiding*

P.M. Session
2:15– 2:45 Chemistry of Anti-thyroid Compounds, with Special Reference to Thiouracil and Hyperthyroidism.
Dr. Astwood.
2:45–3:45 Endocrine Clinic: Treatment of Toxic Goiter.
Dr. Thompson.
3:45– 4:15 Diagnosis and Treatment of Obesity.
Dr. Turner.
4:15– 4:30 Intermission—Refreshments.
4:30– 5:00 Influence of the Adrenals on Tumors of Lymphatic Origin in the Mouse.
Dr. Kendall.
5:00– 5:45 Clinico-Pathologic Conference.
Drs. Sloan and Rukstinat.

Thursday, November 2.

Endocrinology (Continued)

ANTON J. CARLSON, M.D., F.A.C.P., *Presiding*

A.M. Session
8:00– 9:00 Endocrine Clinic: Addison's Disease.
Dr. Thompson.
9:00– 9:30 Control of Corpus Luteum Function.
Dr. Astwood.
9:30–10:00 The Treatment of Coronary Thrombosis.
Dr. Gilbert.

Infectious Diseases

10:00–10:30 Declining Trends and Future Management of Acute Infectious Diseases.
Dr. Hoyne.
10:30–10:45 Intermission—Refreshments.

B. C. H. HARVEY, M.D., *Presiding*

10:45–11:15 Poliomyelitis.
Dr. Levinson.
11:15–11:45 Brucellosis.
Dr. Spink.
11:45–12:45 Methods of Diagnosis in Virus Diseases.
Dr. Francis.
12:45– 1:15 Tularemia.
Dr. Spink.
1:15– 2:15 Luncheon.

Thursday, November 2.

Infectious Diseases (Continued)

ROBERT S. BERGHOFF, M.D., F.A.C.P., *Presiding*

P.M. Session

2:15– 2:45 Review of the Status of the Treatment of Pneumococcus Pneumonia.
Dr. Kelly.

2:45– 3:15 Epidemics of Acute Respiratory Infections due to Hemolytic Streptococci and Their Relation to Rheumatic Fever.
Dr. Spink.

Chemotherapy

3:15– 3:45 Present Status of Sulfonamide Therapy.
Dr. Volini

3:45– 4:15 Penicillin Therapy.
Dr. Spink.

4:15– 4:30 Intermission—Refreshments.

4:30– 5:15 The Treatment of Medical and Surgical Infections with Penicillin.
Lt. Col. Queen.

5:15– 5:45 Treatment of Meningococcus Meningitis with Penicillin.
Capt. McCarthy.

Friday, November 3.

Chemotherapy (Continued)

NEWELL C. GILBERT, M.D., *Presiding*

A.M. Session

8:00– 8:30 Pitfalls and Safeguards in Sulfonamide Therapy.
Dr. Rhoads.

8:30– 9:00 The Mode of Action of Penicillin in Meningococcal and Gonococcal Infections.
Dr. Miller.

9:00– 9:30 Toxic Reactions from Sulfonamide Therapy.
Dr. Blankenhorn.

Hematology

9:30–10:00 The Differential Diagnosis and Treatment of Non-Hemolytic Anemias Resistant to Liver and Iron.
Dr. Doan.

10:00–10:30 Clinical Value of Sternal Puncture.
Dr. Limarzi.

10:30–10:45 Intermission—Refreshments.

10:45–11:15 The Hemolytic Anemias.
Dr. Doan.

11:15–11:45 Diagnosis and Treatment of Leukemia.
Dr. Alt.

11:45–12:15 Indications and Contraindications for the Use of Radioactive Phosphorus Therapy (P 32) in Hematologic States.
Dr. C. V. Moore, Jr.

12:15–12:45 Infectious Mononucleosis.
Dr. Isaacs.

12:45– 1:15 The Significance of Splenomegaly.
Dr. Alt.

1:15– 2:15 Luncheon.

Friday, November 3.

Hematology (Continued)

WILLARD O. THOMPSON, M.D., F.A.C.P., *Presiding*

P.M. Session

2:15– 2:45 The Present Status of Transfusions and Blood Substitutes.
Dr. Doan.

2:45– 3:15 The Rh Factor.
Dr. Davidsohn.

3:15– 3:45 Prothrombin and Its Relation to Bleeding States.
Dr. Quick.

3:45– 4:15 The Pathologic Physiology of Hemorrhagic Diseases.
Dr. C. V. Moore, Jr.

4:15– 4:30 Intermission—Refreshments.

4:30– 5:00 The Rôle of the Spleen in Clinico-Pathologic States.
Dr. Doan.

5:00– 5:30 Non-Tropical Sprue.
Dr. C. V. Moore, Jr.

Saturday, November 4, 1944.

REGIONAL MEETING

OF THE

AMERICAN COLLEGE OF PHYSICIANS

DRAKE HOTEL

East Lake Shore Drive and North Michigan Avenue

PRELIMINARY PROGRAM *

Morning Session

Gold Coast Room

9:00–12:30 Hormones of the Adrenal Cortex.
Dr. Kendall.

The Diagnosis of Beriberi Heart Disease.
Dr. Blankenhorn.

(Title to be announced later.)
Vice Admiral McIntire (or envoy).

New Conceptions in the Therapeutic Use of Exercise.
Major Soto-Hall.

The Treatment of Medical and Surgical Infections with Penicillin.
Lt. Col. Queen.

Penicillin Therapy at the University of Minnesota Hospitals, 1942–44.
Dr. Spink.

Afternoon Session

Ballroom

2:00– 5:00 Rheumatic Fever.
Col. Allen.

Medical Treatment of Hyperthyroidism, with Special Reference to Thiouracil.
Dr. Astwood.

* The final and complete program of the Regional Meeting will be mailed at a later date.

Hepatitis.
Dr. Watson.
Neuroses in the Combat Zone—Mechanism and Prognosis.
Col. Bleckwenn.
Virus Diseases.
Dr. Francis.

Cocktails, 5:30 P.M., Gold Coast Room
Dinner, 7:00 P.M., Gold Coast Room. Short and informal speeches by various distinguished guests.

COURSE NO. 6—SPECIAL MEDICINE

(December 4–15, 1944)

PHILADELPHIA INSTITUTIONS

PHILADELPHIA GENERAL HOSPITAL

34th Street below Spruce

THOMAS M. MCMILLAN, M.D., F.A.C.P., *Director*

(Minimal Registration, 25; Maximal Registration, 50)

This unique program allots for approximately one-half day to the consideration of each of several special fields of medicine. It offers a short but detailed resumé in these several different specialties, and gives an opportunity to study under a faculty selected from various Philadelphia Institutions, including the University of Pennsylvania School of Medicine and Graduate School of Medicine; Jefferson Medical College of Philadelphia; Temple University School of Medicine; the Woman's Medical College of Pennsylvania; the Philadelphia General Hospital; Abington Memorial Hospital; the Children's Hospital; the Institute of the Pennsylvania Hospital; the Pennsylvania Hospital; Presbyterian Hospital; the Jewish Hospital; the Lankenau Hospital and the Department of Public Health of Philadelphia. The faculty is augmented also by several eminent teachers from other cities.

For the convenience of the registrants, a central meeting place, Philadelphia General Hospital, has been selected, rather than having classes held in each individual institution.

The concluding day, Friday, December 15, will be devoted to a Regional Meeting of the American College of Physicians for eastern Pennsylvania, New Jersey, Delaware and adjacent territory. All physicians taking this course, whether they be members of the College or not, are invited to participate in the Regional Meeting, to attend the noon-day luncheon and the evening dinner-meeting.

OFFICERS OF INSTRUCTION

J. Marsh Alesbury, M.D., Chairman of the Philadelphia County Medical Society Committee on Maternal Welfare of the City of Philadelphia.
Frederick H. Allen, M.D., Assistant Professor of Psychiatry, University of Pennsylvania School of Medicine.
Bernard J. Alpers, M.D., Professor of Neurology, Jefferson Medical College of Philadelphia.

Kenneth E. Appel, M.D., F.A.C.P., Assistant Professor of Psychiatry, University of Pennsylvania School of Medicine; Senior Psychiatrist, Institute of the Pennsylvania Hospital.

J. P. Atkins, M.D., Associate in Broncho-esophagology, University of Pennsylvania School of Medicine.

Theodore F. Bach, M.D., F.A.C.P., Associate in Medicine, University of Pennsylvania Graduate School of Medicine.

Oscar V. Batson, M.D., Professor of Anatomy, University of Pennsylvania Graduate School of Medicine.

Joseph T. Beardwood, Jr., M.D., F.A.C.P., Assistant Professor of Medicine, University of Pennsylvania Graduate School of Medicine.

Herman Beerman, M.D., Assistant Professor of Dermatology and Syphilology, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Chief, Outpatient "B" Service, Dermatology and Syphilology, Pennsylvania Hospital.

Moses Behrend, M.D., F.A.C.S., Associate in Surgery, Jefferson Medical College of Philadelphia; Attending Surgeon, Jewish and Mt. Sinai Hospitals; Consulting Surgeon, Department of Thoracic Surgery, Philadelphia General Hospital.

Samuel Bellet, M.D., Instructor in Medicine, University of Pennsylvania School of Medicine; Associate in Cardiology, University of Pennsylvania Graduate School of Medicine; Assistant Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Assistant Chief, Cardiology, Philadelphia General Hospital.

John V. Blady, M.D., Director of the Tumor Clinic, Temple University Hospital.

Marion A. Blankenhorn, M.D., F.A.C.P., Professor of Medicine and Head of the Department, University of Cincinnati College of Medicine, Cincinnati, Ohio.

Harry L. Bockus, M.D., F.A.C.P., Professor of Gastro-enterology, University of Pennsylvania Graduate School of Medicine.

Russell S. Boles, M.D., F.A.C.P., Associate in Medicine, University of Pennsylvania School of Medicine; Gastro-enterologist, Bryn Mawr Hospital.

Earl D. Bond, M.D., Director of Research, Institute of the Pennsylvania Hospital; Professor of Psychiatry and Vice Dean, University of Pennsylvania Graduate School of Medicine.

Ralph S. Bromer, M.D., Clinical Professor of Radiology, University of Pennsylvania Graduate School of Medicine.

Charles L. Brown, M.D., F.A.C.P., Professor of Medicine and Head of the Department of Medicine, Temple University School of Medicine.

W. E. Burnett, M.D., Professor of Surgery, Temple University School of Medicine.

W. Edward Chamberlain, M.D., F.A.C.P., Professor of Radiology, Temple University School of Medicine.

A. Burton Chance, Jr., M.D., Instructor in Orthopedics, University of Pennsylvania Graduate School of Medicine.

Robert Chobot, M.D., F.A.C.P., Assistant Professor of Clinical Pediatrics, New York Post-Graduate Medical School and Hospital, Columbia University; Chief of Pediatric Allergy, New York Post-Graduate Medical School and Hospital; Assistant Chief, Allergy Clinic, Roosevelt Hospital; President, American Academy of Allergy; New York, N. Y.

Frank S. Clarke, M.D., Instructor in Radiology, University of Pennsylvania School of Medicine.

Louis H. Clerf, M.D., F.A.C.P., Professor of Laryngology and Bronchoscopy, Jefferson Medical College of Philadelphia.

Paul C. Colonna, M.D., F.A.C.S., Professor of Orthopedic Surgery, University of Pennsylvania School of Medicine.

Edward S. Dillon, M.D., F.A.C.P., Associate Professor of Diseases of Metabolism, University of Pennsylvania School of Medicine and Graduate School of Medicine; Chief, Metabolic Division, Philadelphia General Hospital.

Charles W. Dunn, M.D., F.A.C.P., Associate in Medicine, University of Pennsylvania Graduate School of Medicine; Endocrinologist, Abington Memorial Hospital.

Thomas M. Durant, M.D., F.A.C.P., Associate Professor of Internal Medicine, Temple University School of Medicine.

W. Wallace Dyer, M.D., F.A.C.P., Instructor in Medicine, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Assistant Chief in Medicine, Philadelphia General Hospital; Physician, Diabetic Outpatient Department, and Staff, Bryn Mawr Hospital.

Mary H. Easby, M.D., F.A.C.P., Associate in Cardiology, University of Pennsylvania Graduate School of Medicine; Chief in Medicine and Chief of Cardiac Clinic, Woman's Hospital.

K. O'Shea Elsom, M.D., Associate in Medicine, University of Pennsylvania School of Medicine.

Gilson C. Engel, M.D., F.A.C.S., Chief, Surgical Service "B", Lankenau Hospital; Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine.

William H. Erb, M.D., F.A.C.S., Associate in Surgery, University of Pennsylvania School of Medicine.

Lowell A. Erf, M.D., F.A.C.P., Associate in Medicine, Assistant Director of the Division of Hematology, and Director of the Transfusion Plasma Unit, Jefferson Medical College of Philadelphia.

George E. Farrar, M.D., F.A.C.P., Associate Professor of Internal Medicine, Temple University School of Medicine.

John T. Farrell, Jr., M.D., F.A.C.P., Clinical Professor of Radiology, University of Pennsylvania Graduate School of Medicine.

Harriet Felton, M.D., Assistant Instructor in Pediatrics, University of Pennsylvania School of Medicine.

Harrison F. Flippin, M.D., F.A.C.P., Assistant Professor of Medicine, University of Pennsylvania Graduate School of Medicine.

George D. Gammon, M.D., F.A.C.P., Associate Professor of Clinical Neurology and Acting Head of the Department of Neurology, University of Pennsylvania School of Medicine.

Leslie N. Gay, M.D., F.A.C.P., Associate in Medicine, Johns Hopkins University School of Medicine; Chief, Protein Clinic, Johns Hopkins Hospital; Baltimore, Md.

Francis C. Grant, M.D., F.A.C.S., Professor of Neurosurgery, University of Pennsylvania Graduate School of Medicine.

J. Q. Griffith, Jr., M.D., F.A.C.P., Associate in Medicine and A. Atwater Kent Fellow in Medicine, University of Pennsylvania School of Medicine.

Paul Gyorgy, M.D., Assistant Research Professor of Pediatrics, University of Pennsylvania School of Medicine.

Samuel B. Hadden, M.D., F.A.C.P., Associate Professor of Neurology, University of Pennsylvania School of Medicine; Visiting Psychiatrist, Philadelphia General Hospital; Neuropsychiatrist, Presbyterian Hospital.

Alice Hamilton, M.D., Consultant, Bureau of Standards, U. S. Department of Labor (formerly Professor of Industrial Toxicology, Harvard School of Hygiene).

Gertrude S. Henle, M.D., Instructor in Pediatrics, University of Pennsylvania School of Medicine.

Werner Henle, M.D., Assistant Professor of Bacteriology in Pediatrics, University of Pennsylvania School of Medicine.

Herman E. Hilleboe, M.D., Medical Director, Chief of Tuberculosis Control Division, U. S. Public Health Service, Washington, D. C.

Joseph F. Hughes, Lt. Comdr., (MC), USNR, U. S. Naval Hospital, Philadelphia; Director of the Laboratories of the Institute of the Pennsylvania Hospital; Neurophysiologist, University of Pennsylvania School of Medicine.

Norman R. Ingraham, Jr., M.D., Chief, Division of Venereal Disease Control, Philadelphia Department of Public Health; Assistant Professor of Dermatology and Syphilology, University of Pennsylvania School of Medicine; Chief, Syphilis Clinic, and Consultant, Philadelphia General Hospital.

Harold W. Jones, M.D., F.A.C.P., Thomas Drake Cardeza Professor of Clinical Medicine and Hematology, Jefferson Medical College of Philadelphia; Director, Charlotte Drake Cardeza Foundation and Laboratories of the Division of Hematology.

Joseph V. Klauder, M.D., Associate Professor of Dermatology and Syphilology, University of Pennsylvania Graduate School of Medicine; Director, Ocular Syphilis Clinic, Wills Hospital.

Paul Klemperer, M.D., Pathologist, Mt. Sinai Hospital, New York, N. Y.

John A. Kolmer, M.D., F.A.C.P., Professor of Medicine, Temple University School of Medicine; Director, Research Institute of Cutaneous Medicine; Consultant in Serology, U. S. Public Health Service.

John Lansbury, M.D., F.A.C.P., Associate Professor of Medicine, Temple University School of Medicine.

William G. Leaman, Jr., M.D., F.A.C.P., Professor of Medicine, Woman's Medical College of Pennsylvania.

Samuel A. Loewenberg, M.D., F.A.C.P., Clinical Professor of Medicine, Jefferson Medical College of Philadelphia; Physician, Philadelphia General Hospital.

Francis D. W. Lukens, M.D., Assistant Professor of Medicine and Director of the George S. Cox Medical Research Institute, University of Pennsylvania School of Medicine.

Elizabeth P. Maris, M.D., Instructor in Pediatrics, University of Pennsylvania School of Medicine.

Albert A. Martucci, M.D., Director of the Department of Physical Medicine, Abington Memorial Hospital.

Robert A. Matthews, M.D., Associate Professor of Psychiatry, Jefferson Medical College of Philadelphia; Psychiatrist, Institute of the Pennsylvania Hospital.

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Thomas M. McMillan, M.D., F.A.C.P., Associate Professor of Cardiology, University of Pennsylvania Graduate School of Medicine.

Franklin R. Miller, M.D., Assistant Director, Division of Hematology, and Associate Professor of Medicine, Jefferson Medical College of Philadelphia.

Merle M. Miller, M.D., F.A.C.P., Associate in Allergy, University of Pennsylvania Graduate School of Medicine; Chief of Allergy Clinic, Graduate Hospital of the University of Pennsylvania.

T. Grier Miller, M.D., F.A.C.P., Professor of Clinical Medicine, University of Pennsylvania School of Medicine.

Sarah I. Morris, M.D., F.A.C.P., Professor of Preventive Medicine, Woman's Medical College of Pennsylvania.

F. L. Munro, Ph.D., Research Chemist, Division of Hematology, Jefferson Medical College and Hospital.

Muriel P. Munro, Ph.D., Research Chemist, Division of Hematology, Jefferson Medical College and Hospital.

Meyer Naide, M.D., Instructor in Medicine, University of Pennsylvania School of Medicine.

John R. Neefe, Capt., (MC), AUS, Assistant Instructor in Medicine, University of Pennsylvania School of Medicine.

Waldo E. Nelson, M.D., Professor of Pediatrics, Temple University School of Medicine.

Josef B. Nylin, M.D., Associate in Physiotherapy, University of Pennsylvania School of Medicine.

A. M. Ornsteen, M.D., F.A.C.P., Assistant Professor of Neurology, University of Pennsylvania School of Medicine; Neurologist, Hospital of the University of Pennsylvania, Philadelphia General and Jewish Hospitals.

Harold D. Palmer, M.D., F.A.C.P., Professor of Psychiatry, Woman's Medical College of Pennsylvania; Associate Professor of Psychiatry, University of Pennsylvania School of Medicine; Senior Psychiatrist, Institute of the Pennsylvania Hospital.

Ethel G. Peirce, M.D., Assistant Visiting Physician, Service of Rheumatoid Diseases, Abington Memorial Hospital.

Ralph Pemberton, M.D., F.A.C.P., Professor of Medicine, University of Pennsylvania Graduate School of Medicine; National Consultant in Rheumatism and Arthritis, War-Time Graduate Medical Meetings.

Eugene P. Pendergrass, M.D., F.A.C.P., Professor of Radiology, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Director, Department of Radiology, Hospital of the University of Pennsylvania.

William Harvey Perkins, M.D., F.A.C.P., Dean and Professor of Preventive Medicine, Jefferson Medical College of Philadelphia.

George Morris Piersol, M.D., F.A.C.P., Director, Center for Research and Instruction in Physical Medicine, University of Pennsylvania.

Alison H. Price, M.D., Assistant Demonstrator in Medicine, Jefferson Medical College of Philadelphia.

Milton Rapoport, M.D., Associate in Pediatrics, University of Pennsylvania School of Medicine.

Rufus S. Reeves, M.D., F.A.C.P., Director, Philadelphia Department of Public Health.

Horace Reider, M.D., Chief Medical Resident, Bryn Mawr Hospital.

Hobart A. Reimann, M.D., Magee Professor of Practice of Medicine and Clinical Medicine, Jefferson Medical College of Philadelphia.

Stanley P. Reimann, M.D., F.A.C.P., Associate Professor of Surgical Pathology, University of Pennsylvania Graduate School of Medicine; Pathologist and Director of Research Institute, Lankenau Hospital.

John G. Reinhold, Ph.D., Principal Biochemist, Philadelphia General Hospital.

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Leonard G. Rowntree, M.D., F.A.C.P., Colonel, (MC), AUS, Director, Philadelphia Institute for Medical Research; Chief of Medical Division, Selective Service, Washington, D. C.

Mitchell I. Rubin, M.D., Associate Professor of Clinical Pediatrics, University of Pennsylvania School of Medicine.

David A. Sampson, M.D., Associate in Radiology, University of Pennsylvania Graduate School of Medicine.

William G. Sawitz, M.D., Assistant Professor of Parasitology, Jefferson Medical College of Philadelphia.

William H. Schmidt, M.D., Director of the Department of Physical Medicine, Jefferson Medical College of Philadelphia.

Truman G. Schnabel, M.D., F.A.C.P., Professor of Clinical Medicine, University of Pennsylvania School of Medicine; Physician, Philadelphia General and Presbyterian Hospitals.

Mildred W. Schram, Ph.D., Secretary, International Cancer Research Foundation.

C. Wesler Scull, Ph.D., Instructor in Chemistry (Assigned to Medicine), University of Pennsylvania Graduate School of Medicine.

Murray J. Shear, Ph.D., Biochemist, National Cancer Institute, Bethesda, Md.

Will Cook Spain, M.D., F.A.C.P., Clinical Professor of Medicine, New York Post-Graduate Medical School and Hospital, Columbia University; Chief of Allergy Clinic, New York Post-Graduate Medical School and Hospital; New York, N. Y.

John H. Stokes, M.D., Professor of Dermatology and Syphilology, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Director, Institute for the Control of Syphilis, University of Pennsylvania.

Joseph Stokes, Jr. M.D., William H. Bennett Professor of Pediatrics, University of Pennsylvania School of Medicine.

John Stouffer, M.D., Director, Psychopathic Department, Philadelphia General Hospital.

Edward A. Strecker, M.D., F.A.C.P., Professor of Psychiatry, University of Pennsylvania School of Medicine; Consultant-in-Chief, Institute of the Pennsylvania Hospital; Special Consultant in Psychiatry to the Surgeon General of the Navy and to the Secretary of War for the Army and the Army Air Forces.

William D. Stroud, M.D., F.A.C.P., Professor of Cardiology, University of Pennsylvania Graduate School of Medicine; Cardiologist, Pennsylvania Hospital; Cardiologist and Director of Heart Station, Bryn Mawr Hospital; Physician-in-Chief, Cardiovascular Service, Abington Memorial Hospital.

Paul C. Swenson, M.D., Professor of Radiology, Jefferson Medical College of Philadelphia.

Leandro M. Tocantins, M.D., Assistant Director, Division of Hematology, and Associate Professor of Medicine, Jefferson Medical College of Philadelphia.

James G. Townsend, M.D., Chief, Industrial Hygiene Division, U. S. Public Health Service, Bethesda, Md.

Louis N. Tuft, M.D., Assistant Professor of Medicine and Chief of Allergy Clinic, Temple University School of Medicine.

Henry J. Tumen, M.D., F.A.C.P., Assistant Professor of Medicine, University of Pennsylvania Graduate School of Medicine.

D. L. Turner, Ph.D., Associate in Chemistry and Research Chemist to the Division of Hematology, Jefferson Medical College and Hospital.

Elizabeth M. Turner, Ph.D., Research Chemist to the Division of Hematology, Jefferson Medical College and Hospital.

Louis H. Twyeffort, M.D., Instructor in Psychiatry, University of Pennsylvania School of Medicine.

Jacob H. Vastine, M.D., Professor of Radiology, Woman's Medical College of Pennsylvania.

C. Richard Walmer, M.D., Westinghouse Electric and Mfg. Co.

Matthew Walzer, M.D., Associate in Medicine, Cornell University Medical College; Attending in Allergy and Chief of Allergy Clinic, Jewish Hospital, Brooklyn, N. Y.

Virgene Wammock, M.D., Assistant Director, Institute for the Control of Syphilis, University of Pennsylvania.

W. L. White, M.D., Harrison Fellow in Surgery, University of Pennsylvania School of Medicine.

Bernard P. Widmann, M.D., Professor of Radiology, University of Pennsylvania Graduate School of Medicine.

DeForest P. Willard, M.D., Professor of Orthopedics, University of Pennsylvania Graduate School of Medicine.

N. W. Winkelman, M.D., Professor of Neuropathology, University of Pennsylvania Graduate School of Medicine; Medical Director, Philadelphia Psychiatric Hospital.

Thomas H. Wright, M.D., Instructor in Psychiatry, University of Pennsylvania School of Medicine; Clinical Director, Department for Mental and Nervous Diseases, Pennsylvania Hospital.

H. A. Zintel, M.D., Instructor in Surgery, University of Pennsylvania School of Medicine.

OUTLINE OF COURSE

Monday, December 4.

Gastro-enterology

T. GRIER MILLER, M.D., F.A.C.P., *In Charge*

A.M. Session

9:00–12:00
1. Etiology and Pathogenesis of Cirrhosis of the Liver.
   Dr. Tumen.
2. Management of Cirrhosis of the Liver (Clinic).
   Dr. Brown.
3. Some Studies on Hepatitis in Volunteers.
   Drs. Neefe and Reinhold.
4. Emotional Aspects of Gastrointestinal Disease.
   Dr. Twyeffort.
5. The Non-Surgical Abdomen (Clinic).
   Dr. Schnabel.
6. Present Status of Regional Ileitis (Clinic).
   Dr. Bockus.
7. Peptic Ulcer (Clinic).
   Dr. Boles.

Monday, December 4.

Chemotherapy

HARRISON F. FLIPPIN, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00– 5:00 Panel Discussion: Chemotherapy.

1. Clinical Significance of Sulfamerazine Blood and Spinal Fluid Levels.
   Dr. Reinhold.
2. Local Use of Sulfonamides and Penicillin in Surgical Infections.
   Dr. Zintel.
3. Local Use of Penicillin in Bronchopulmonary Infections.
   Dr. Atkins.
4. Prophylactic Use of Penicillin in Chest Surgery.
   Dr. Burnett.
5. Treatment of Empyema and Meningitis with Penicillin.
   Dr. White.
6. Subacute Bacterial Endocarditis Treated with Penicillin.
   Dr. Mayock.
7. Para-aminohippuric Acid—Clinical Studies.
   Dr. Flippin.

Tuesday, December 5.

Pediatrics

JOSEPH STOKES, JR., M.D., *In Charge*

A.M. Session

9:00–12:00
1. The Influence of Sulfonamide Therapy on the Course of Acute Glomerulonephritis.
   Drs. Rubin and Rapoport.

2. The Present Status of Methods of Preventing Epidemic Influenza.
   Drs. Werner Henle, Gertrude Henle and Stokes.
3. A Reliable Skin Test for Determination of Susceptibility to Pertussis.
   Dr. Felton.
4. Why and How the Proper Prevention of Psychoneuroses Starts in Childhood.
   Dr. Allen.
5. Nutritional Factors in Ectoparasitic Infections with Special Reference to Infestation with Lice.
   Dr. Gyorgy.
6. The Value of Susceptibility Tests for Mumps.
   Dr. Maris.

Tuesday, December 5.

Vitamin Deficiency

THOMAS M. DURANT, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00– 5:00
1. Ascorbic Acid.
   Dr. Durant.
2. Vitamin D.
   Dr. Nelson.
3. Vitamin K.
   Dr. Farrar.
4. Thiamin.
   Dr. Elsom.
5. Pellagra.
   Dr. Blankenhorn.

Wednesday, December 6.

Heart Disease

WILLIAM G. LEAMAN, JR., M.D., F.A.C.P., *In Charge*

A.M. Session

9:00–12:00
1. Treatment of Cardiac Emergencies.
   Dr. Bellet.
2. Congestive Cardiac Failure.
   Dr. Leaman.
3. Peripheral Failure.
   Dr. Durant.
4. Hypertension.
   Dr. Griffith.
5. Treatment of Acute Rheumatic Carditis.
   Dr. Easby.
6. Coronary Artery Disease.
   Dr. Stroud.

Wednesday, December 6.

Metabolic Diseases

EDWARD S. DILLON, M.D., F.A.C.P., *In Charge*

P.M. Session.

2:00– 5:00
1. Pathogenesis of Diabetes Mellitus.
   Dr. Lukens.

2. Surgical Aspects of Diabetic Gangrene.
   Dr. Erb.
3. The Standardization of Diabetes Mellitus by Diet and Insulin.
   Dr. Beardwood.
4. Biochemical Aspects of Diabetic Coma.
   Dr. Reinhold.
5. The Use of Plasma in the Treatment of Diabetic Coma.
   Drs. Dyer and Reider.
6. Frequent Errors in the Diagnosis of Diabetes Mellitus.
   Dr. Dillon.

Thursday, December 7.

Arthritis and Related Conditions

RALPH PEMBERTON, M.D., F.A.C.P., *In Charge*

A.M. Session
9:00–12:00

Symposium

1. Statistical Factors.
2. Pathology.
3. Physiologic Disturbances Involved.
4. Clinical Presentation of Cases, with Emphasis on Diagnostic Methods and Treatment.
5. Round Table Discussion.
   Drs. Pemberton, Bach, Scull and Peirce.

Thursday, December 7.

Physical Medicine

GEORGE MORRIS PIERSOL, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00 The Present Obligations and Opportunities of Physical Medicine.
Dr. Piersol.

2:20 Rôle of Physical Medicine in the Problem of Low Back Pain.
Dr. Colonna.

2:40 Physiotherapy in the Control of Neuromuscular Pain.
Dr. Martucci.

3:00 Nerve Palsies, Diagnosis and Prognosis (Moving Pictures).
Dr. Batson.

3:20 The Influence of Posture on Disease.
Dr. Schmidt.

3:40 Diathermy: Its Uses and Abuses.
Dr. Nylin.

4:00 Value of Physical Therapy in Peripheral Vascular Disease.
Dr. Naide.

4:20 Physical Medicine in the Management of Poliomyelitis.
(a) Modern Treatment of the Acute Stage.
Dr. Chance.
(b) The Rôle of the Techniques of Physical Medicine in the Chronic Stage.
Dr. Willard.

Friday, December 8.

Hematology

HAROLD W. JONES, M.D., F.A.C.P., *In Charge*

A.M. Session

9:00–12:00

1. Treatment of Polycythemia.
   Dr. Erf.
2. Observations on the Anticephalin Activity of Normal and Hemophilic Plasmas.
   Dr. Tocantins.
3. The Influence of Myeloid and Lymphoid Stimulating Substances on Two Mouse Tumors.
   Dr. Franklin R. Miller.
4. Electrophoretic Separation of Factors Involved in Blood Coagulation.
   Dr. Muriel P. Munro.
5. The Chemistry of Myeloid and Lymphoid Stimulating Substances.
   Dr. D. L. Turner.
6. Properties of an Anti-Coagulant Present in the Blood of a Hemophiliac.
   Dr. F. L. Munro.
7. Cellular Infiltrations of Organs of Guinea Pigs that Receive Extracts of Livers and Spleens of Patients Dead with Leukemia.
   Dr. Erf.
8. Plasma Anticephalin Activity in Hemorrhagic and Thrombotic Disorders.
   Dr. Tocantins.
9. The Influence of Myeloid and Lymphoid Stimulating Substances on Tissue Culture of Fibroblasts.
   Dr. Elizabeth M. Turner.
10. Ultraviolet-irradiated Blood Transfusion as a Therapeutic Measure.
    Dr. Jones.

Friday, December 8.

Allergy

MERLE M. MILLER, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00– 2:20 Laboratory Procedures in the Diagnosis of Allergic Diseases. Skin Testing.
Dr. Tuft.

2:20– 3:00 Pathology of Bronchial Asthma.
Dr. Klemperer.

3:00– 3:30 Diagnosis and Treatment of Bronchial Asthma.
Dr. Gay.

3:30– 4:00 Seasonal Pollinosis. Diagnosis and Treatment.
Dr. Walzer.

4:00– 4:20 Allergy in Children. Rôle of Heredity.
Dr. Chobot.

4:20– 4:40 Extracts: Methods of Preparation and Standardization.
Dr. Spain.

4:40– 5:00 Demonstration of Skin Testing. Passive Transfer.
Dr. Miller.

Saturday, December 9.

Acute Infectious Diseases

HOBART A. REIMANN, M.D., *In Charge*

A.M. Session

9:00–12:00 1. Penicillin in Meningitis.
Dr. Price.
2. Acute Infectious Diarrheal Diseases.
Dr. Reimann.
3. Host Types in Tuberculosis Infections.
Dr. Perkins.
4. Malaria.
Dr. Sawitz.

Monday, December 11.

THE BROAD ASPECTS OF PUBLIC HEALTH

RUFUS S. REEVES, M.D., F.A.C.P., *In Charge*

A.M. Session

9:00 Public Health Aspects of Maternal and Child Welfare.
Dr. Alesbury.
9:30 Rehabilitation Problem of the Neuro-psychiatric Service Men.
Dr. Strecker.
10:00 Rheumatic Fever from the Standpoint of Public Health.
Dr. Stroud.
10:30 Newer Techniques in Tuberculosis Control.
Dr. Hilleboe.
11:00 Recess.
11:15 Round Table Discussion.

Monday, December 11.

Industrial Medicine

SARAH I. MORRIS, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00– 5:00 1. Industrial Medicine's Challenge to the General Practitioner.
Dr. Townsend.
2. Various Types of Industrial Poisons: Their Mode of Entrance and Action.
Dr. Hamilton.
3. The Pitfalls of Diagnoses in Lead Poisoning.
Dr. Walmer.

Tuesday, December 12.

Psychiatry

HAROLD D. PALMER, M.D., F.A.C.P., *In Charge*

A.M. Session

9:00–12:00 1. Introductory Remarks.
Dr. Strecker.
2. Electroencephalographic Diagnosis.
Dr. Hughes.

3. Electric Shock Therapy in Psychiatry.
   Dr. Wright.
4. The Problem of the Depressed Patient.
   Dr. Matthews.
5. The Rewards of Illness.
   Dr. Bond.
6. Psychotherapy in Medical Practice.
   Dr. Appel.
7. Clinical Demonstration of Psychiatric Cases.
   Dr. Stouffer.

Tuesday, December 12.

Neurology

A. M. ORNSTEEN, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00– 5:00
1. Functional Traumatic Neuropsychiatric Syndromes.
   Dr. Ornsteen.
2. The Pathogenesis of Cerebral Aneurysms.
   Dr. Alpers.
3. The Present Status of the Intervertebral Disk.
   Dr. Grant.
4. Penicillin Therapy for Neurosyphilis.
   Dr. Gammon.
5. Migraine and Its Equivalents.
   Dr. Winkelman.

Wednesday, December 13.

Endocrinology

CHARLES W. DUNN, M.D., F.A.C.P., *In Charge*

A.M. Session

9:00–12:00
1. Thyroid.
   Dr. Rose.
2. Adrenals.
   Dr. Lukens.
3. Hypopituitary States.
   Dr. Lansbury.
4. Hyperpituitary States.
   Dr. Loewenberg.
5. Endocrine Disorders in Selective Service Cases.
   Dr. Rowntree.
6. Male Hormone Therapy.
   Dr. Dunn.

Wednesday, December 13.

Syphilis

NORMAN R. INGRAHAM, JR., M.D., *In Charge*

P.M. Session

2:00 Introductory Remarks. The War as an Impelling Influence in the Control of Syphilis.
Dr. Ingraham.

2:15 The Problem of Falsely Positive and Doubtful Serologic Reactions in the Diagnosis of Syphilis (Lecture, Lantern Slide Demonstration and Discussion).
Dr. Kolmer.

2:50 Blindness Caused by Syphilis with a Statement on the Present Status of Penicillin Therapy in Ocular Syphilis (Lantern Slide Demonstration and Illustrative Case Records).
Dr. Klauder.

3:25 Penicillin in the Treatment of Early and Late Syphilis (Clinic and and Discussion).
Drs. John H. Stokes, Beerman and Wammock.

4:20 Early Diagnosis and Non-Specific Measures in the Treatment of Neurosyphilis (Clinic and Discussion).
Dr. Hadden.

Thursday, December 14.

Tumors

STANLEY P. REIMANN, M.D., F.A.C.P., *In Charge*

A.M. Session

9:00–12:00
1. Organization and Results of Anti-Cancer Examination Clinics in Philadelphia.
Dr. Schram.
2. Chemicals as Inciters of Malignant Growths.
Dr. Shear.
3. Carcinoma of the Colon.
Dr. Behrend.
4. Carcinoma of the Stomach.
Dr. Engel.
5. Pulmonary Carcinoma.
Dr. Clerf.
6. Carcinoma of the Mouth.
Dr. Blady.

Thursday, December 14.

Roentgenology

EUGENE P. PENDERGRASS, M.D., F.A.C.P., *In Charge*

P.M. Session

2:00– 5:00
1. The General Use of X-Rays in Obstetrics.
Dr. Swenson.
2. Roentgenologic Consideration of Inflammatory Conditions of the Chest.
Dr. Clarke.
3. The Roentgenology of the Upper Cervical Spine and Base of the Skull.
Dr. Chamberlain.
4. The Roentgen Diagnosis of Gastrointestinal Conditions in Infants and Children.
Dr. Bromer.
5. The Roentgenologic Problems of the Stomach and Duodenum.
Dr. Widmann.
6. The Roentgenologic Problems of the Colon.
Dr. Farrell.

7. Sialography.
   Dr. Blady.
8. Subcutaneous Urography.
   Dr. Vastine.
9. The Roentgenology of the Urinary Tract.
   Dr. Sampson.

Friday, December 15.

REGIONAL MEETING

OF THE

AMERICAN COLLEGE OF PHYSICIANS

Morning Session

WILLIAM HARVEY PERKINS, M.D., F.A.C.P., *In Charge*

JEFFERSON MEDICAL COLLEGE HOSPITAL

1025 Walnut Street

A.M. Session

9:00–12:00 The Staff of the Jefferson Medical College and Hospital will present a program of clinics and demonstrations, details of which will be included in a special Regional Meeting Program that will be published later and placed in the hands of each registrant in advance of the opening of this course.

P.M. Session

1:00 Buffet Luncheon.
College Headquarters, 4200 Pine Street.

Afternoon General Session

WILLIAM D. STROUD, M.D., F.A.C.P., *In Charge*

BENJAMIN FRANKLIN HOTEL

9th and Chestnut Streets

Ballroom, Mezzanine Floor

2:00– 5:00 A Symposium on Rheumatic Fever by eminent authorities. Detailed Program will be furnished to each registrant in advance of the opening of this course.

6:30 Cocktail Party (Betsy Ross Room) and Dinner Meeting (Grand Ballroom).

Guests will include Regents and Officers of the College, the Surgeons General, or their official envoys, of the U. S. Army, U. S. Navy and U. S. Public Health Service, and other distinguished medical men. Selected, timely, short addresses will be given by the President of the College, Dr. Ernest E. Irons, and others.

READING LIST AND BIBLIOGRAPHY

An attempt is made to obtain reading lists for each Postgraduate Course for publication in the ANNALS OF INTERNAL MEDICINE, making these lists available to the entire membership of the College, in addition to preparing better the men who will take the courses. These lists are not to be considered as all-inclusive.

Allergy—Course No. 4

Textbooks

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Asthma and Hay Fever in Theory and Practice. A. F. Coca, M. Walzer and A. A. Thommen. Charles C. Thomas, Baltimore, 1931.

Clinical Allergy. Louis Tuft. W. B. Saunders Co., Philadelphia, 1937.

Occupational Diseases of the Skin. Louis Schwartz and Louis Tulipan. Lea and Febiger, Philadelphia, 1939.

Monographs

Allergy. C. E. Von Pirquet. Archives of Internal Medicine *7:* 259, 1911.

Anaphylaxis, Hypersensitiveness and Allergy. W. W. C. Topley. An Outline of Immunity, Chapter 12, p. 192. Wm. Wood Co., 1935.

Hypersensitiveness, Anaphylaxis, Allergy. H. Gideon Wells. The Chemical Aspects of Immunity, Chapter 9, p. 225, second edition. Chemical Catalog Co., New York, 1929.

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Articles

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Serological Evidence of Immunity with Co-existing Sensitization in a Type of Human Allergy. Hay Fever. R. A. Cooke, J. H. Barnard, S. Hebald and A. Stull. J. Exper. Med. *62:* 773, 1935.

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Studies in the Transmission of Sensitization from Mother to Child in Human Beings. S. D. Bell and Z. Eriksson. J. Immunol. *20:* 447, 1931.

The Placental Transmission of Antibodies in the Skin-Sensitive Type of Human Allergy. W. B. Sherman, S. F. Hampton and R. A. Cooke. J. Exper. Med. *72:* 611, 1940.

The Question of the Elimination of Foreign Protein (Eggwhite) in Woman's Milk. H. H. Donnally. J. Immunol. *19:* 15, 1930.

The Production in the Rabbit of Hypersensitive Reactions to Lens, Rabbit Muscle and Low Ragweed Extracts by the Action of Staphylococcus Toxin. E. L. Burky. J. Allergy *5:* 466, 1934.

General Clinical Allergy

History Taking in Allergic Diseases. F. M. Rackemann. J. A. M. A. *106:* 976, 1936.

Studies in Specific Hypersensitiveness. III. On Constitutional Reactions: The Dangers of the Diagnostic Cutaneous Test and Therapeutic Injection of Allergens. R. A. Cooke. J. Immunol. *7:* 119, 1922.

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Asthma

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